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SYNOPSIS OF SURGERY

Stanley Stillman

A SYNOPSIS OF SURGERY

By ERNEST W. HEY GROVES,
M.S., M.D., B.Sc. (Lond.), F.R.C.S. (Eng.)
Assistant Surgeon to the Bristol General Hospital;
Surgeon to the Cossham Hospital;
Senior Demonstrator of Anatomy at the University College, Bristol.

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PREFACE

The title of this small book describes its aim and scope. In recent years the tendency of all surgical literature to grow in bulk and diversity has of necessity compelled authors to enlarge their text-books more and more. Complete treatises on surgery occupy many volumes, and even the most concise require months for their perusal, so that a student often despairs of being able to revise his knowledge.

The present volume is an attempt to make an epitome of the salient facts in surgical practice, and to place these facts in such a manner that they may most easily and rapidly be referred to or revised. It has been compiled almost entirely from notes made by the author in preparing students for examinations.

It is of course obvious that the large works cannot be dispensed with; but after a student has carefully read through a complete text-book, which ought to be done at the time he is actually engaged in ward and out-patient work, he may find this synopsis aid his memory in retaining a vast array of facts in an orderly manner.

The busy practitioner, who is expected to keep in practical touch with an ever advancing science, has too often to allow the big books to go unread, whilst magazine articles are disconnected, discursive, and difficult for reference. It is thought that this synopsis may, by its methodical arrangement, prove of assistance by presenting the diagnosis and treatment of surgical conditions in a concise manner.

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The special arrangement of headings, type, and indented margins enables the reader to see the scope of the subject at a glance, and to refer to any part of it at will. It may also offer some suggestions as to the arrangement of answers in a clear and succinct manner at examinations.

In the case of certain parts of the body where diagnosis is most difficult, e.g., the bones, joints, abdomen, and inguino-scrotal region, sections have been inserted dealing with the systematic examination of these parts.

Only the main points of operations are referred to, so that the principles which underlie each procedure may not be lost in a mass of details.

Anatomical and pathological facts are only inserted when they are essential to a comprehension of clinical signs and operative measures, but a summary of surface markings is added in the last chapter, which includes all those of importance.

In the main arrangement of the book, and in the sequence of the chapters, that most comprehensive and clear of all text-books, "Rose and Carless," has been followed, and where special treatises or monographs are quoted, the reference is given in a foot-note.

E. W. H. G.

Clifton, July, 1908.

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SYNOPSIS OF SURGERY.

CHAPTER I.

SURGICAL BACTERIOLOGY.

General Characters.—FISSION FUNGI—DEVOID OF CHLOROPHYL—UNICELLULAR VEGETABLE ORGANISMS—CONSIST OF PROTOPLASM SURROUNDED BY A CELL WALL.

Reproduction.

(*a*) BY SIMPLE FISSION—the common method.
(*b*) BY SPORE FORMATION—only found in a few bacilli.
Never found in common pyogenic bacteria.
Occurs, e.g., in *B. anthracis*, *B. tetani*, *B. œdematis maligni*.
Spores are formed when conditions for bacterial life are unfavourable. They have a thick capsule and are very strongly resistant to desiccation, heat, or antiseptics.

Locomotion.

PASSIVE.—By Brownian movement or physical or chemical currents.

ACTIVE.—By flagellæ, i.e., actively contractile protoplasmic processes.
Found in *B. typhosus* and *B. coli*.

Shape.

COCCI. Round; never produce spores.
Staphylococci: arranged irregularly.
Diplococci: arranged in pairs.
Streptococci: arranged in chains.
Tetracocci: arranged at 4 corners of a square.
Sarcinæ: arranged at 8 corners of a cube.
BACILLI. Rod-shaped, often spore-bearing.
Streptobacilli: chains of bacilli.
Leptothrix: long threads.
SPIRILLA. Spirally curved.

Physiology.

THEY REQUIRE ORGANIC PROTEIDS for food because they have no chlorophyl.
Parasites obtain it from living animals or plants.
Saprophytes obtain it from dead tissues.
Facultative parasites ordinarily live on dead tissues, but can also grow in living, e.g., *B. proteus*.
Facultative saprophytes ordinarily live in living tissues, but can with difficulty be cultivated outside, e.g., gonococci.

Physiology, *continued.*

CONDITIONS NECESSARY for bacterial existence or activity :—

Proteid food in an assimilable form—Water—Temperature at or about blood heat—Darkness—Oxygen or its absence.

Aërobes, e.g., pyogenic cocci, require oxygen.

Anaërobes, e.g., *B. tetani*, cannot live in oxygen. But they can often live in company with other bacteria which absorb oxygen even in the presence of air ; e.g., *B. tetani* can grow in a surface wound if it is contaminated by cocci.

CONDITIONS CAUSING A SUSPENSION of bacterial activity or growth.

Absence of pabulum—Dryness—Cold—Sunlight.

CONDITIONS CAUSING BACTERIAL DEATH.

Heat at or below boiling point quickly kills all except spores. Spores require temp. of 250° F. or long boiling to kill.

Chemical reagents, e.g., carbolic acid or mercury salts.

Must be of a certain strength—Act for a certain time—Come into intimate contact.

Contact with living tissues.

In all conditions in which the living tissues are more potent than the bacteria.

PRODUCTS OF BACTERIAL ACTIVITY when growing in living tissues.

Acids or alkalies, e.g., *B. coli* produces an acid.

Gases (rare), e.g., *B. coli* or *B. œdematis maligni.*

Pigments (rare), e.g., *B. pyocyaneus.*

Aromatics—indol, phenol. Alcohols.

Ferments—diastase, curdling ferment, proteolytic ferment.

Ptomaines—crystallizable alkaloids.

Toxins.—The specific poisons, probably albumoses.

(*a*) Intracellular toxins, e.g., tuberculosis. The toxin remains closely associated with the bacillus, and only acts in its neighbourhood.

(*b*) Extracellular toxins, e.g., diphtheria, tetanus. The toxin is found in the body fluids, and acts at a distance from the bacteria.

Theories of Immunity.

THE TISSUES ACQUIRE TWO METHODS OF DEFENCE.

1. They kill the bacteria—antibacterial action.
2. They neutralize the toxins—antitoxic action.

ANTITOXIC ACTION is an actual chemical antithesis to the toxin, produced in the blood by infection.

This is a proteid, and is called antitoxin ; it can be made to neutralize the toxin outside the body.

Best exemplified in diphtheria and tetanus.

ANTIBACTERIAL ACTION is brought about by two agencies :—

1. Phagocytosis. The leucocytes digest the bacteria.
2. Action of antibacterial substances in the serum which kill and dissolve the bacteria outside the leucocytes.

BACTERIOLYSINS. Exemplified in antistreptococcus, anti-cholera, anti-plague, anti-pneumococcus, and anti-typhoid sera.

PHAGOCYTOSIS. Is increased by the action of a substance called an opsonin, produced by the action of the bacteria and toxins in the blood.

OPSONIC INDEX is the proportion of bacteria devoured by leucocytes in an infected blood to those devoured by leucocytes in normal blood.

Directly after injection of a vaccin (bacteria or toxins):—

The opsonic index falls = negative opsonic phase. During this time a further dose of vaccin does harm.

After a few days:—

The opsonic index rises = positive opsonic phase. During this time a further dose of vaccin increases the phagocytosis.

Distribution.

IN AIR AND WATER. Plentiful in proportion to warmth and proximity to organic life.

IN EARTH. Always abundant near the surface.

IN ANIMALS (apart from disease).

On and in the skin, hair follicles, and sweat glands.

Mouth, especially round the teeth.

Whole alimentary canal. Most numerous at the end of the small and the beginning of the large intestine.

Lower parts of the urethra, vagina, and nose.

Infection.—The invasion of the living tissues by bacteria in such a manner as to produce local or general disease.

CONDITIONS NECESSARY FOR INFECTION.

1. DOSE OF BACTERIA.—Bacteria must be introduced in a certain number, or they will be destroyed before they produce any effect.
2. VIRULENCE.—This varies enormously naturally.
   May be increased artificially: By passage through successive animals.
   May be decreased artificially: By cultivation outside the body, especially if done under unfavourable conditions.
3. PATHOGENICITY.—Only certain kinds of bacteria are capable of producing disease in certain kinds of animals.
4. SUSCEPTIBILITY OF THE HOST,—i.e., a vulnerability of the living tissues by the bacteria.
   Increased by: Any constitutional depression, cold or wet, starvation, youth of the patient, alcoholism and other narcotics, constitutional diseases, e.g., Bright's or diabetes.
5. ENTRANCE OF THE BACTERIA into the tissues, provided by:—(*a*) A wound or abrasion or ulcer; (*b*) An inflamed or injured part, e.g., an inflamed tonsil or a sprained joint; (*c*) Profound constitutional depression, allowing the normally present bacteria to enter and multiply in the tissues.

Immunity.—Is a condition of absolute or relative insusceptibility of the individual to infection after invasion by virulent pathogenic bacteria. There are two varieties:—

Natural Immunity. Racial or individual.

Acquired Immunity. Active immunity; passive immunity.

NATURAL IMMUNITY.—Nature of the condition is not known.

No antitoxic or antibacterial substance can be separated from the blood.

Is lessened by: Cold or wet, starvation, youth, hæmorrhage, alcohol or poisons.

ACTIVE IMMUNITY.—Caused by a previous infection, either as the natural or modified disease.

Involves an actual illness—Takes time for its development—Lasts many years.

Is brought about by: (*a*) Previous attack; (*b*) Giving the disease, e.g., old-fashioned inoculation of small-pox; (*c*) Injection of attenuated virus, e.g., vaccination for small-pox, anthrax (in cattle), hydrophobia; (*d*) Injection of dead bacteria, e.g., plague, typhoid, tuberculosis (T.R.); (*e*) Injection of toxins, e.g., in the animals which provide the antitoxins of diphtheria and tetanus.

PASSIVE IMMUNITY.—Caused by injecting the blood of an actively-immunized animal—Involves no illness—Follows immediately on injection—Only lasts a few months.

Is most successful against extracellular toxins, e.g., diphtheria and tetanus.

CHAPTER II.

INFLAMMATION AND SUPPURATION.

INFLAMMATION.

Definition.—The changes that follow injury of living tissues.

Causes: (1) Mechanical injuries; (2) Bacterial invasion; (3) Thermal injuries—heat or cold; (4) Chemical injuries; (5) Electrical injuries—high-tension currents—X rays.

Histological Changes.

I.—VASCULAR CHANGES.

HYPERÆMIA.—The small vessels dilate—The blood-stream slows after momentary acceleration.

STASIS.—The blood-stream comes to a standstill—The blood clots in the vessels.

EXUDATION.—The white cells, which have been hugging the vascular wall, now creep through it—The red cells escape passively from the vessel—The serum of the blood oozes out from the vessel.

II.—TISSUE CHANGES.—Vary with the nature and virulence of the infective agent, with the resistance of the individual, and with the local circulatory conditions.

May be one of three processes, viz.:—

(*a*) PROLIFERATION.—Results from mild or chronic infections. The connective and endothelial cells multiply by division. Fresh connective tissue is formed thereby.
Produces thickening, fibrosis or sclerosis.

(*b*) GRANULATION.—Produced by the non-pyogenic bacteria. Exuded lymphocytes form a mass of "small round cells," which invades and replaces the tissues.

(*c*) DESTRUCTION.

(i.) Colliquative necrosis. The result of non-bacterial injury, especially burns.
Excessive fluid exudation, with separation and disintegration of cells, forms a blister.

(ii.) Coagulation necrosis. The result of infection by pyogenic organisms.
The exuded serum containing bacteria and toxins coagulates round the tissue cells. Many of the tissue cells and leucocytes are killed by the toxins.
Ends in one of four ways.

1. Catarrh.—The dead cells are thrown off from a free mucous surface, but only the superficial parts are lost.

Histological Changes, *continued.*

2. Ulceration.—The dead cells are thrown off from a free surface in such numbers as to leave an area devoid of skin or mucous membrane.
3. Suppuration.—The coagulated tissue is peptonized and liquefied by the bacteria and leucocytes. Polynuclear leucocytes destroy and are destroyed by the bacteria.
4. Sloughing or gangrene. The tissue dies *en masse* and forms a dead slough.

Local Results.

I.—RESOLUTION without any permanent effect; only possible when the process has stopped short of thrombosis.

The circulation is resumed.

The exuded cells and serum are absorbed.

II.—ORGANIZATION of the inflammatory products.

Only occurs when the infective agent is mild.

The granulation tissue or "small round cells" and the connective tissue cells form fibroblasts, and these form new connective tissue.

New vessels are formed by a budding out from old vessels.

III.—DESTRUCTION of the tissues, usually followed by repair by granulation tissue.

ULCER.—The loss of tissue on a free surface.

ABSCESS.—The liquefied dead tissues below the surface.

SLOUGH.—A piece of tissue which has been killed *en masse*, but not liquefied.

GANGRENE.—The death of tissues in massive quantity. Usually applied to the death of an organ or part of a limb.

N.B.—Ulceration, sloughing, or gangrene may be caused by other things besides inflammatory infections.

Signs.

1. Heat } caused by hyperæmia.
2. Redness }
3. Swelling, caused by hyperæmia and exudation.
4. Pain, increased by pressure or movement.
5. Loss of function.

Symptoms.—Febrile symptoms are nearly always present, but they are due to the infection and not to the inflammation. They vary according to the nature of the infection and resistance.

Treatment of Acute Inflammation.

LOCAL.

Remove the exciting cause if possible. Rest the inflamed part.

Cold applications to prevent hyperæmia.

Only suitable in non-toxic cases, e.g., sprained joints.

ENCOURAGEMENT OF HYPERÆMIA to hasten natural process: Hot fomentations.

BIER'S PASSIVE HYPERÆMIA* in acute inflammation is applied in three ways. (1) By an elastic bandage tied round a limb or round the neck at a distance from the inflamed focus. This should be tight enough to obstruct the venous return without affecting the arterial pulse, and should relieve, not increase, the pain. It is left on for twenty hours out of twenty-four at first, and for shorter periods later. (2) By cupping-glasses, which are exhausted by an attached rubber ball or suction-pump. These are placed over the affected focus when situated on the trunk. They are applied for about one hour a day, the suction being released every five minutes for one minute. (3) Large glass receptacles into which the whole or part of a limb can be placed and the air partly exhausted. In all cases the treatment does not obviate the necessity for early incisions directly suppuration has occurred, but these incisions should be only short, small openings, and it is not necessary to pack them.

---

* Schmieden and Meyer on "Bier's Hyperæmia."

DEPLETION OF BLOOD: Leeches — Scarification — Multiple incisions.

CONSTITUTIONAL.

Rest in bed. Low diet.
Antitoxic sera to neutralize the toxins.
Antibacterial sera to kill the bacteria.
Inoculation with modified toxins to increase opsonins.

Treatment of Chronic Inflammation.

Remove the cause if possible. Rest.

ACTIVE HYPERÆMIA: Massage—Hot-air treatment.

COUNTER-IRRITATION: Blister—Seton—Actual cautery—Iodine or mercurial applications.

PRESSURE: Bandaging—Elastic pressure—Scott's dressing—Unna's paste.

SUPPURATION.

Definition.—The molecular necrosis and liquefaction of living tissues.

Varieties.

RATE OF DEVELOPMENT.—Acute or chronic.

NATURE OF INFECTION.—Simple pyogenic infection.

Specific infection with the bacteria of a specific fever, e.g., typhoid, tubercle.

SITUATION.—

MUCOUS MEMBRANE: Catarrh, e.g., acute otitis media.

SEROUS MEMBRANE: Purulent serositis, e.g., peritonitis or empyema.

FREE SURFACES: Ulceration—"Inflamed ulcers."

BELOW THE SURFACE: Abscess—localized suppuration; Cellulitis—diffuse suppuration.

Cause.—Infection by bacteria.
Through a wound.
Through the lymph or blood-stream.

Bacteria which cause suppuration.

I.—SIMPLE PYOGENIC BACTERIA, i.e., those which producc suppuration without causing any specific fever.

1. Staphylococcus Pyogenes: Aureus, Albus, and Citreus.
   - Grow readily in culture media.
   - Form luxuriant patches in a few days.
   - Form a peptonizing ferment and therefore liquefy gelatin.
   - Form clusters of minute dots in the growth or tissues.
   - Are the commonest cause of suppuration.
   - The aureus is the most virulent. The albus is the least virulent.
   - Common cause of :—
     - Boils, superficial abscesses; Carbuncles (here a slough is produced ; *vide infra*)—Peritonitis—Empyema—Osteomyelitis and arthritis.
2. Streptococcus Pyogenes.
   - Forms chains of minute dots.
   - Grows slowly outside the body.
   - Forms minute dot-like colonies.
   - Does not produce a peptonizing ferment, and therefore does not liquefy gelatin.
   - Causes Virulent suppuration ; Erysipelas—Cellulitis—Acute arthritis — Peritonitis — Empyema — Septicæmia and pyæmia.
3. Bacillus Coli Communis.
   - Occurs in the healthy intestine.
   - Is a motile bacillus with flagella.
   - Grows readily in culture media.
   - Produces Characteristic odour—Free gas—Acid.
   - Causes Acute peritonitis—Cystitis. Any abdominal suppuration—Rarely abscesses elsewhere in bones, joints, or soft parts.
4. Bacillus Pyocyaneus.
   - Grows in wound surfaces—Produces greeny-blue pus—Very mild in its toxic action.

II.—SPECIFIC PYOGENIC BACTERIA.—Bacteria which produce a well-marked specific disease, and occasionally local suppuration.

1. Pneumococcus.
   - Is a diplococcus. Usually encapsuled.
   - Grows outside the body with difficulty.
   - Is the ordinary cause of pneumonia.
   - Also causes :—
     - Empyema: common. Peritonitis, Arthritis, Osteomyelitis: rare.
2. Bacillus Typhosus.
   - A multiflagellate bacillus.
   - Is the ordinary cause of enteric fever.
   - May cause :—
     - Bone suppurations—periostitis. Suppuration in the gall-bladder. Local peritonitis.
3. Gonococcus (*vide infra*).
4. Tubercle.

Minute Anatomy of an Abscess.

I.—IN THE EARLY STAGES it shows all the signs of acute inflammation, ending in coagulation necrosis.

II.—IN THE FULLY MATURED ABSCESS the central portion has become liquid pus.

The wall from without inwards consists of four zones :—

(1) HYPERÆMIA and exudation.
(2) THROMBOSIS with round-celled infiltration.
(3) "SMALL ROUND CELLS" with many bacteria.
(4) Central collection of PUS.

III.—IN THE HEALING ABSCESS a zone of fibrosis intervenes between the granulation tissue and the healthy tissue.

The innermost layers consist of granulation tissue, i.e., small round-celled tissue permeated by new blood-vessels.

Bacteria are practically absent, or dead.

Nature of Pus.—FLUID of sp. gr. 1030.

Contains 85% water.—Opaque white or yellow colour—Alkaline in reaction.

The liquid portion consists of a solution of peptones.

The solid portion consists of: Dead polynuclear leucocytes—Living leucocytes—Bacteria—Débris of partially digested tissues.

Signs and Symptoms of Acute Suppuration.

SIGNS OF ACUTE INFLAMMATION, and especially :—

FLUCTUATION—showing the fluidity of contents.
ŒDEMA of the skin—of great value in deep suppuration.
HARD BRAWNY TEXTURE.
PAIN becoming distinctly THROBBING.

GENERAL SIGNS are those of a bacterial infection, and especially :

RIGOR or shivering fit, with rapid rise of temperature.

LEUCOCYTOSIS. Leucocytes increase from 5,000 to anything up to 100,000 per cmm.

Absent in severe fatal infections.
It indicates local infection well resisted.

Treatment of Suppuration.

I.—IN EARLY STAGES when suppuration is doubtful, treat as for acute inflammation.

II.—WHEN PUS IS CLEARLY INDICATED :—

Open freely from the surface.
Open all secondary loculi into the main cavity.
Make the opening in the most dependent position.
Make as many openings as are necessary to freely drain the cavity.
Swab out the pus or irrigate with antiseptics.
Do not scrape the wall, for fear of infecting fresh tissues.
Put on an antiseptic dressing. Drain with tubes or gauze.
Continue fomentations or passive hyperæmia.

Chronic Abscess.

DEFINITION. A collection of pus without the signs of inflammation. Usually of very slow development.

PATHOLOGY.

TUBERCLE is the cause in the vast majority of cases.

PYOGENIC COCCI may give rise to an abscess, and then the organisms die or become latent, and the abscess remains cold and chronic. Especially in connection with bones.

ANATOMY.

May be connected with any tuberculous focus.

Commonly connected with BONE, JOINT, or LYMPHATIC disease.

Ceases to be a chronic abscess when it is infected with active pyogenic organisms. Hence the predilection for sites removed from skin or mucous membrane.

GROWTH : Slowly spreads along lines of areolar tissue. Guided by fascial planes, or the sheaths of blood-vessels.

WALL : Usually thick and well defined. Lined by a layer of soft granulations which forms " the pyogenic membrane." These contain tubercle bacilli and giant cell systems which form caseous masses in the tubercular cases. A dense layer of fibrocicatricial tissue lies outside the granulations.

CONTENTS : Pus of a special character. Curdy and fatty débris. Very few cells, and these degenerate. Occasionally cholestrine crystals. No recognizable bacteria. Pus is usually infective to animals.

COMMON EXAMPLES of Chronic Tuberculous Abscesses.

PSOAS ABSCESS. Caused by dorsi-lumbar tuberculous disease.

Spreads beneath the internal arcuate ligament. Guided by the sheath of the psoas muscle. Forms a fluctuating swelling above Poupart's ligament on the outer side of vessels. Dips beneath the femoral vessels, and enters the thigh behind and on the inner side. May continue down the thigh to the knee.

Open over the fluctuating spots. Usually :—

(*a*) Above the outer half of Poupart's ligament, or
(*b*) Below the inner half of Poupart's ligament, or
(*c*) In the loin through the quadratus lumborum.

Occasionally at two of these places.

ILIAC ABSCESS. Arises from disease of the ilium, sacro-iliac joint, or of the hip joint through the acetabulum. Forms a fluctuating swelling in the iliac fossa. Open above outer half of Poupart's ligament.

LUMBAR ABSCESS. Usually caused by disease of the lumbar vertebræ. Is directed by the dorsal branches of the lumbar arteries. Tracks beneath the psoas and on inner side of quadratus. Points on outer side of the erector spinæ.

Open by a vertical incision over the outer border of erector spinæ.

COSTAL OR INTERCOSTAL ABSCESS. Caused by disease of the ribs or of the dorsal or cervical vertebræ.

Open over the fluctuating swelling.

CHRONIC RETROPHARYNGEAL ABSCESS. Caused by disease of the upper cervical vertebræ.
  Lies behind the prevertebral fascia.
  Open behind the sternomastoid.

TERMINATION OF CHRONIC ABSCESS.

1. RECOVERY by absorption of the abscess contents.
    - The contents become caseous, cretified, or are absorbed.
    - A fibrous scar remains.
    - Seldom happens except in very small abscesses.
2. BURSTING externally or into a mucous canal.
    - Rapidly infected with pyogenic organisms.
    - Incurable or fatal sepsis results.
3. DEATH FROM SEPTIC ABSORPTION after pyogenic infection.
    - Usually of a hectic type.
4. DEATH FROM AMYLOID DISEASE after pyogenic infection.
    - Waxy degeneration occurs in the vessels and viscera, viz.:
        - In the arterioles and capillaries. In the liver, spleen, kidneys, and intestines.
    - Reactions of the waxy substance.
        - Iodine—turns brown. Sulphuric acid after iodine—inky blue. Methyl violet—red.
    - Symptoms and signs.
        - Enlargement of the liver and spleen. Albuminuria, with great increase of amount of urine, with casts. Diarrhœa.
    - Condition can be arrested if the radical removal of the suppurating focus is done early.
5. RECURRENCE after spontaneous absorption or after operation is very common.

SYMPTOMS AND SIGNS.

- Swelling of slow appearance.
- Absence of heat, redness, tenderness, or raised temperature.
- Fluctuation is well marked unless the abscess is very deep.
- Other symptoms and signs are merely those of the cause of the abscess, e.g. :—
    - Signs of caries of the spine or of sacro-iliac disease.
    - Sepsis, acute or chronic, only develops after pyogenic infection, i.e., after the abscess has opened in the majority of cases.
    - Amyloid disease.

TREATMENT.

- Open the abscess with rigid antiseptic precautions.
- Evacuate the contents.
- Gently scrape the walls as far as possible with a flushing spoon, irrigating with sterilized water.
- Seek for and remove any loose sequestra of bone.
- Leave about one drachm of iodoform in the cavity.
- Sew up without drainage.

CHAPTER III.

SINUS, FISTULA, & ULCERS.

Definition of Sinus and Fistula.—A Sinus is a track lined by granulations, leading from a free surface into the depths of the tissues. A Fistula is a track which opens at both ends on to an external or internal cutaneous or mucous surface.

Structure.—An outer layer of fibrous tissue, which in old cases becomes very thick and dense like cartilage. An inner layer of granulations, usually infected by bacteria.

Causes.—Abscess—Penetrating wounds.

CAUSES OF NON-CLOSURE of sinuses and fistulæ.

- Dead septic body in the sinus, e.g., necrosed bone.
- Foreign body which is septic, e.g., ligature.
- Insufficient opening of a deep cavity.
- Inability of a deep cavity to collapse, e.g., empyema.
- Tuberculous or septic infection of the walls of the sinus.
- Constant passage of septic discharges, e.g., fæcal or urinary fistula.
- Massive growth of fibrous tissue like cartilage round the sinus.
- Growth of epithelium down the sinus.

Treatment of Sinus or Fistula.

- Lay it freely open.
- Provide efficient drainage or close altogether the cavity it leads to.
- Scrape away all granulations from its walls.
- Remove any septic bodies.
- Swab with pure carbolic in very septic cases.
- Excise the whole track where possible when it is very dense.
- Pack the cavity left, and allow to heal by granulation.

SPECIAL TREATMENT OF FISTULA.

- Special plastic operations to close the inner opening, or other special operations.

Definition of Ulcer and Ulceration.—An Ulcer is a superficial loss of tissue involving a part of the skin or mucous membrane. Ulceration is a molecular necrosis of superficial tissues leading to a loss of substance.

Classification by their cause.

I.—TRAUMATIC. Caused by injury.

Mechanical: the results of wounds. Chemical: the results of caustics. Thermal: the results of burns. Electrical: the results of electric currents or X rays.

II.—PYOGENIC. Caused by inflammation due to the ordinary pyogenic cocci.

III.—CONGESTIVE. A reduced vitality due to deficient circulation is the chief cause, but traumatism and pyogenic infection always play their part.

IV.—NECROTIC. Due to loss of tissue by sloughing. This may be septic or aseptic.

V.—TROPHIC. Due chiefly to nerve lesions causing anæsthesia.

VI.—SPECIFIC. Due to special bacterial diseases, e.g., tubercle, syphilis, anthrax.

VII.—MALIGNANT. Due to pressure of a malignant growth, or to a replacement of skin or mucous membrane by new growth, which then breaks down by ulceration.

General Features of Ulcers. Points to be noticed:—

1. SURFACE.
   - Usually depressed below the healthy surface.
   - Elevated above the surrounding surface in the following:—Ulcer with exuberant granulations; malignant or any fungating growth.
   - Sloughy, smooth, granulating, or fungating, according to the stage of ulceration.
   - Granulations may be vascular, e.g., in healing ulcers; œdematous, e.g., in tubercular; fibrous, non-vascular, in syphilitic.
2. EDGE.
   - Eroded and dark red or covered with slough: when the ulceration is actively spreading.
   - Rounded, smooth, thick edge: when the ulcer is stationary.
   - Shelving, with white, blue, and red zones: healing.
   - Thick, infiltrated, raised, or everted: new growth.
   - Clean cut, "punched out": especially in syphilitic tertiary ulcers.
   - Gyrate, i.e., made up of parts of several smaller circles: usually tertiary syphilis.
   - Undermined, when an ulcer follows a subcutaneous growth abscess, or gumma: often seen in tubercle, sarcoma, syphilis.
   - Surrounded by mass of hard epidermis: perforating ulcer.
3. RELATION TO STRUCTURES BENEATH.
   - Moveable over deep structure: healthy and healing.
   - Fixed to deep structures: chronic, stationary.
   - Fixed to deep tumour: new growth.
   - Penetrating deeply into bones and joints: perforating ulcer.
4. DISCHARGE.
   - Purulent and free, containing active bacteria: in spreading cases.
   - Scanty and serous: in healing cases.
   - Bleeds readily and copiously: malignant.
   - Bleeds moderately: healthy granulations.
   - Does not bleed though granulating: syphilis.

General Features of Ulcers, ***continued.***

Leaves chalky deposit : gouty.
May contain special structures, e.g., actinomycosis granules, epitheliomatous cells, etc.

5. CONDITION OF THE SURROUNDING TISSUES.
   Swollen, œdematous, congested : congestive ulcers.
   Inflamed : pyogenic ulcers.
   Quite healthy, with abrupt transition : tertiary syphilis.
   Deep induration : malignant.
6. POSITION.
   Traumatic ulcers may be anywhere, most commonly on exposed situations.
   Congestive ulcers : lower half of leg.
   Tubercular ulcers : in neck, groins, or axillæ, i.e., over glands or over joints. May occur anywhere.
   Lupus : face (nose, mouth, ears), fingers or toes.
   Syphilis (tertiary) : may be anywhere, but especially near the knees, buttocks, outer side of thighs. Also in face, nose, and mouth.
   Malignant : anywhere, but most commonly in mouth and tongue, breast, external genitals.
   Trophic or Perforating : Site of any old scar or ulcer. Beneath the sole of the foot, usually under the great toe.
7. SHAPE.
   Simple congestive ulcers tend to be round or oval.
   Tertiary specific ulcers may be circinate, i.e., formed by the coalescence of several ulcers or islands of skin ; may be present in the midst of an ulcer from the same cause.
8. NUMBER.
   Multiple ulcers, especially if widely scattered, indicate some constitutional infection, e.g., tubercle, secondary syphilis.
   Multiple ulcers all on the same part indicate some local infection by pyogenic bacteria.
   Single large ulcers are either of mechanical, congestive, malignant, or tertiary syphilitic origin.
9. RATE OF PROGRESS.
   Short history : rapid healing is found in most simple pyogenic ulcers.
   Extreme chronicity : especially in congestive ulcers, also in rodent ulcers.
   Steady growth : malignant ulcers.
10. MICROSCOPICAL CHARACTER of the growing edge.
    Shows malignant or tubercular character.

Stages of Simple Ulceration.

I.—EXTENSION. The inflamed ulcer.

STRUCTURE. Is that of the wall of an acute abscess.

CHARACTERS.

Surface : Covered with sloughs or lymph. No granulations.
Discharge : Copious, thin, offensive.

Margins : Acutely inflamed, with well-defined edge.
Base : Thick and adherent.

TREATMENT : Rest—Fomentations—Passive hyperæmia.

II.—STATIONARY OR CHRONIC STAGE. The stage between tissue necrosis and tissue formation, i.e., between sloughing and granulation. Theoretically every ulcer undergoes this stage, but only in chronic ulcers is it well marked. In healthy ulcers tissue regeneration begins directly necrosis is at an end.

STRUCTURE. The inflammatory zones of hyperæmia and exudation become converted into fibro-cicatricial layers.

The surface is covered by a layer of small, round cells, not sufficiently vascularized to form granulations.

CHARACTERS.

Surface : Smooth, glazed, and shiny.
Discharge : Scanty and serous.
Margins : May be congested, but are not inflamed.
Edges : Rounded, hard, and elevated.
Base : Often adherent to underlying structures, e.g., the periosteum.
Skin around is very congested, and often pigmented with marked œdema in " congestive " cases.

CAUSES OF CHRONICITY, i.e., of non-healing of an ulcer.

1. Defective circulation : In lower parts of legs—In those who stand all day—In old people.
2. Venous obstruction : Varicose veins—After venous thrombosis, e.g., " white leg "—Pregnancy.
3. Fixation of the ulcer to underlying bone or fascia : Prevents the edges being drawn together—Prevents the base being drawn up to the edges—Prevents the proper vascularization of the base of the ulcer necessary for the formation of granulations.
4. Constitutional conditions : Bright's disease—Gout—Diabetes, etc.
5. Continued irritation : When an ulcer is left dirty and without any dressing.
6. Pressure of œdema. This causes the vessels in the neighbourhood to be occluded, and produces an extension of the ulcer.

VARIETIES OF CHRONIC SIMPLE ULCERS.

1. Varicose ulcer. Due to congestion of varicose veins of the leg.
2. Congestive ulcer. Due to congestion of the leg from weak circulation, long standing, etc.
3. Eczematous ulcer. Superficial, not deeper than the dermis. Copious discharge.
4. Irritable ulcer. Over the ankle. Small but very painful. Exposes terminal nerve filaments.
5. Ulcer after a burn or other traumatism. When the whole skin thickness is destroyed, and when a larg ulcer overlies a dense fascia or bone. Adhesions of t base to this prevent cicatricial contraction.

Stages of Simple Ulceration, *continued.*

- Complications of chronic simple ulcers.
  - Œdema of the parts below, from the pressure of cicatricial contraction on the blood- and lymph-vessels.
  - Periostitis of the underlying bone, forming a large periosteal node.
  - Epithelioma: The edges become thickened, everted, or warty—A very slow growing form of cancer, which may remain for years—Lymphatic extension is very slow, because the lymph channels have been destroyed.
- Treatment of Chronic Simple Ulcers.
  - Posture: Rest with elevation of the foot.
  - Pressure: Simple or elastic bandages—Starch bandages or Unna's paste over dressings—Strapping.
  - Dressings:
    - When the surface is foul: Charcoal or boracic fomentations.
    - For mere indolence: Metal, e.g., a piece of pewter cut to the size and shape of the ulcer—Metallic ointments, especially zinc—Resins, e.g., resin ointment—Oxygen, applied for ½ hour daily.
    - Method: If ointments are used they should be spread on lint cut to the shape but rather smaller than the ulcer. All old ointment should be cleaned off daily with oil.
  - Operative Treatment.
    - Multiple incisions in a longitudinal direction to relieve congestive œdema.
    - Undercutting the ulcer edges so as to free them from deep adhesions.
    - Tying prominent varicose veins above the ulcer.
    - Skin grafting. Most useful in traumatic, e.g., burn ulcers.
    - Amputation: For old ulcers of large size with base fixed to the periosteum—For ulcers encircling more than half the circumference of the limb and producing great œdema of the parts below—For epithelioma.

III.—STAGE OF REPAIR.

- Structure.
  - The floor is covered with small round cells grouped round new vascular loops, thus forming the granulations.
  - When the cavity is filled with granulations, the epidermis grows over them from the edge, and so covers the surface.
  - The granulation tissue becomes converted into fibrous tissue, the deeper layers undergoing this change first.
  - The contraction of this fibrous tissue is essential to the repair of all large or deep ulcers.
    - (1) It draws the base up to the edges.
    - (2) It draws the edges together.

CHARACTERS.

Surface : Covered by red granulations.

Edge : Shelves gradually into the ulcer and is on the same level with the granulations. Shows a white, blue, and red margin.

White margin = heaped-up epidermis.

Blue zone = granulations seen through thin, transparent epidermis.

Red zone = granulations.

Base is free from the underlying tissues, i.e., movable over them.

Margins are free from inflammation or congestion.

TREATMENT OF A HEALING SIMPLE ULCER.

Protection by simple dressing.

Prevent the granulations sprouting up above the skin margins by silver nitrate.

Trophic or Perforating Ulcer of the Foot.

CAUSES.

PREDISPOSING.—Diseases of the cord, e.g., tabes or syringomyelia; neuritis, whether diabetic, syphilitic or alcoholic; peripheral nerve lesions causing anæsthesia.

EXCITING.—Pressure of a corn or an abrasion which is unnoticed and neglected because of the anæsthesia.

CHARACTERS.—The orifice is on the sole of the foot, usually under the ball of the great toe. The margins are formed by heaped-up hard epithelium.

A long sinus leads into the deep tissue of the foot, usually involving carious bones and disorganized joints.

It may have acquired a second opening on the dorsum of the foot (hence the term perforating ulcer).

Probing is conspicuously painless.

TREATMENT.—Rest in bed. Freely opening up and scraping out the unhealthy tissues. Cutting away all the exuberant epidermis. Subsequent protection from pressure by suitable padding.

Amputation may be necessitated if many of the tarsal bones and joints are diseased.

CHAPTER IV.

GANGRENE.

Definition.—Death of the tissues *en masse*.

Usually applied when the whole or a part of a limb or viscus is affected, with all the constituent tissues.

Signs.

(1) CESSATION OF CIRCULATION: Loss of pulse—Vessels do not empty on pressure. (2) LOSS OF HEAT. (3) LOSS OF SENSATION. (4) LOSS OF FUNCTION. (5) CHANGE OF COLOUR: Usually dark.

All these signs may be present and yet *recovery may take place* under the following conditions:—Small area affected—Absence of the conditions favourable for sepsis—Good circulation in the neighbourhood.

In certain conditions the above signs indicate *inevitable gangrene*:—

When all the vessels going to a part are affected, i.e., when terminal vessels are concerned, e.g., terminal parts of a limb or testis with rotated cord.

When there are conditions favourable for sepsis, e.g., strangulated gut.

Further signs which prove that death has occurred:—(6) MUMMIFICATION or drying of the tissues. (7) DECOMPOSITION of the tissues. (8) LINE OF ULCERATION between the dead and living tissues.

Varieties.

I.—DRY GANGRENE. Death followed by mummification.

(*a*) PRIMARILY DRY. Caused by blocking of the arteries whilst the veins remain patent, thus draining the tissues of fluid.

(*b*) SECONDARILY DRY. Caused by a loss of fluids from the tissues by evaporation in a case of moist gangrene which has remained aseptic.

The affected parts become hard, dry, and shrivelled.

Liberated hæmoglobin makes the tissues black.

Liberated fat causes some transparency of the skin, through which the deep tissues can be seen.

Sepsis is either absent, or unable in the absence of moisture to produce decomposition.

Terminates by an ulcerating line of demarcation which separates the mass, chiefly at the expense of the dead tissues.

It has little or no tendency to spread.

If extension occurs it is due to the original cause of the gangrene acting on tissues higher up.

Hence extension is rare, and when it occurs it is usually by leaps, not a gradual, steady process.

Associated with very slight febrile symptoms.

Usually is extremely painful.

Rarely causes death; but death may be hastened by the pain and want of sleep.

II.—MOIST GANGRENE. Gangrene of blood-filled tissues.

Veins as well as arteries are blocked, or congestion occurs simultaneously with arterial blocking.

(*a*) It may become dry (very rarely) if asepsis be maintained until evaporation can take place.

(*b*) It usually leads to rapid *putrefaction*. Hence in the vast majority of cases

Moist gangrene = death + putrefaction.

The affected parts are purple and swollen.

Superficial tissues are raised in blisters.

Deep tissues become emphysematous from the gases of decomposition.

Adjacent living tissues are much inflamed.

Terminates by a broad zone of ulceration which separates the mass at the expense of the living tissues.

It spreads steadily and swiftly by infection and thrombosis of adjacent living tissues.

Associated with marked toxæmic and febrile symptoms.

Pain is great, but is dulled by the toxæmia.

Causes death from septicæmia or secondary hæmorrhage.

The Tissue Changes that follow Gangrene.—These consist in a reaction between the dead and living tissues.

Three factors are concerned.

1. The living tissues actively attack the dead tissues.
2. The dead tissues are removed partly by molecular and partly by massive necrosis.
3. Bacteria in the dead tissues attack and destroy some of the living tissues.

The actual result will depend on the relation between these three factors, and the dead tissues will suffer one of three fates.

I.—ABSORPTION.

This really means their disintegration and removal by molecular necrosis.

The living tissues are healthy and vascular.

Sepsis is absent or insignificant.

The amount of dead tissue is small.

The living tissue produces granulation tissue.

The granulations erode and digest the dead tissue.

II.—SEPARATION BY SEPTIC ULCERATION.

The bulk of the dead tissue is large.

The Tissue Changes that follow Gangrene, *continued.*

Or the vitality and circulation in the living tissues weak.

The granulating zone cannot penetrate far from its own base.

It merely corrodes the adjacent zone of dead tissue until it is completely separated.

The separated dead mass drops off or remains as a sequestrum.

III.—SEPARATION BY SEPTIC ULCERATION.

The dead tissues contain virulent bacteria.

Acute septic inflammation is caused in the living tissues thereby.

A zone of living tissue undergoes molecular necrosis, i.e., it suppurates.

Thus the line of separation between living and dead tissues forms at the expense of the living.

The final demarcation of the living tissue by a granulating zone is distinctly higher up than that of the original gangrene.

The process is one of acute suppuration.

Varieties of Gangrene in relation to its cause.

I.—SYMPTOMATIC. Gangrene caused by some vascular or constitutional condition.

(1) Embolic. (2) Senile. (3) Thrombotic. (4) Diabetic. (5) Raynaud's. (6) Ergot.

II.—TRAUMATIC.

(1) Direct: when an injury kills the part injured.

(2) Indirect: when an injury kills a part at a distance from the injury by damage of main vessels.

III.—THERMAL. A variety of direct traumatic gangrene.

(1) Burns. (2) Frost-bite.

IV.—INFECTIVE. Gangrene resulting from septic inflammation.

(1) Acute inflammatory gangrene. (2) Necrosis of bone. (3) Cancrum oris. (4) Carbuncle.

Embolic Gangrene.

EMBOLUS is dislodged from diseased cardiac valves. Or from a dilated left ventricle. Or from an atheromatous plate.

PREDISPOSING CONDITIONS are

Weak circulation } In cases of endocarditis.
Infective organisms in embolus }

Diseased vessels with collaterals unable to dilate, in arterio-sclerosis and atheroma.

SITE OF IMPACTION OF THE EMBOLUS.—At the bifurcation of vessels, e.g., when the Popliteal divides into ant. and post. tibials—Femoral divides into superficial and deep—Brachial divides into radial and ulnar.

SPECIAL FEATURES.

PAIN: Sudden, severe, and lasting.

DRY GANGRENE appears in the extremities, e.g., the toes, especially in asthenic, anæmic people—Affects only the terminal vessels of the blocked trunk—Its extent gives no indication of the position of block—Usually limited by a joint.

MOIST GANGRENE: In a full-blooded patient—Or as a result of secondary venous thrombosis.

DRY AND MOIST GANGRENE is common, e.g., dry gangrene of toes with moist gangrene spreading up the calf.

Senile Gangrene.

PREDISPOSING CONDITIONS.—Calcareous arteries whose collaterals cannot dilate—Weak circulation—Anæmia or some constitutional disease, e.g., nephritis.

EXCITING CAUSE.—Thrombosis in the arteries, often determined by some injury or inflammation, or cold.

SYMPTOMS.

Premonitory: Cold feet, numbness, and cramps.
Dry gangrene beginning in one toe, usually the great toe.
With or without inflammation at its origin.
The gangrene has all the signs of the dry variety (*vide supra*).
Intense pain throughout.

Thrombotic Gangrene apart from traumatism is very rare.

Occurs after typhoid and other long cases of toxæmia.
It is usually in the leg, affecting the femoral.
Dry gangrene follows simple arterial thrombosis usually.

Diabetic Gangrene.—There are three distinct factors in its causation.

1. A BLOOD CONDITION favourable to septic processes.
2. An OBLITERATING ARTERITIS, especially in the tibials.
3. NEURITIS, with some posterior sclerosis of the cord.

MOIST GANGRENE in its worst form is the common type.

Elderly patients—Chronic diabetes—Begins as a whitlow or suppurating corn—Rapidly spreads and is very fatal—Is very likely to give rise to diabetic coma.

DRY GANGRENE usually results from arterial sclerosis and neuritis—It is much less painful than other forms of dry gangrene—When neuritis is a chief cause it may be quite anæsthetic.

Raynaud's Disease.

Young patients—Generally women.

Associated with angioneurotic œdema, paroxysmal hæmoglobinuria, and other neuroses.

Affects fingers, toes, ears, or nose, in superficial patches, symmetrically on both sides of the body.

Of slow development, producing anæmia, cyanosis, and then a superficial slough.

Very painful.

Treat by galvanism or faradism.

Direct Traumatic Gangrene.

The parts which die are at the seat of injury.

The gangrene is caused by a lesion of the blood-vessels locally, thrombosis, obliteration by pressure or rupture.

Trophic disturbances and sepsis may take a large part in the causation.

Direct Traumatic Gangrene, *continued.*

I.—CRUSHED LIMBS. (*See* " Contused Wounds.")

II.—SPLINT PRESSURE: Too tight bandaging or ill-fitting or ill-padded splint—Usually over a bony prominence—Caused by a pressure-obliteration of vessels—Affects the superficial tissues over a bone.

III.—BED-SORES occur over bony points on which a bedridden patient lies—Any prominent vertebral spine—Sacrum or posterior iliac spines—Trochanters—Heels.

Especially in condition of Emaciation—Debility of fevers, e.g., enteric—Any spinal cord lesion, especially traumatic paraplegia.

Trophic Disturbance is the most important factor in spinal cases. The sores are then apt to be acute in their origin and progress.

Septic Discharges—e.g., urine and fæces—also often play an important part in the causation.

Condition.—Superficial skin slough, limited to points of pressure in most cases—Deep spreading sloughing in acute cases with spinal cord lesions.

Prevention.—Careful nursing, i.e., Smooth clean sheets—Constant cleansing from urine or fæces—Keeping the back as dry as possible (washing with spirit after soap and water, dusting with boracic powder)—Use of water bed or air cushions to prevent localized pressure on the prominent bony points.

Treatment.—General cleanliness.
Stimulating ointments, e.g., resin or Friar's balsam.

IV.—ACTION OF CORROSIVE CHEMICALS.

Strong acids, e.g., sulphuric or nitric acids—Strong alkalies, e.g., potash, carbolic acid.

Course, varieties, and treatment are the same as in burns; but with acids and alkalies neutralization should be attempted if the injury is quite recent, so as to stop the corrosive action spreading.

Indirect Traumatic Gangrene.—Caused by an injury obstructing the vessels. The parts that die are at a distance from the injury.

I.—LIGATURE OR THROMBOSIS OF MAIN ARTERY when the circulation is too weak or the vessels too diseased to carry on the collateral circulation.

Similar to embolic gangrene.

II.—OBSTRUCTION OF BOTH THE MAIN ARTERY AND VEIN.—By ligature or injury—By tight bandaging—By strangulation of an internal organ—Primary rupture of an artery and secondary pressure on veins.

Moist gangrene occurs up to the site of obstruction.

Infective Gangrene.—Caused by the direct toxic effect of micro-organisms.

I.—LOCALIZED GANGRENE. Usually staphylococcal.

Here a dense condition of the tissues, e.g., dense bone or subcutaneous fascia, determines the gangrene because the vessels become strangulated by inflammatory exudation in an unyielding matrix.

1. NECROSIS OF BONE (*vide infra*).
2. CARBUNCLE OR BOIL OF THE SKIN (*vide infra*).

II.—SPREADING GANGRENE.

1. CANCRUM ORIS AND NOMA.—One attacks the mouth, the other the genitals—Affects weakly children after exanthemata —Chiefly streptococcal—Slough begins on the mucous membrane, and spreads right through to the skin—Destroys the whole cheek down to the bone.
   - Produces: sapræmia, septicæmia, or septic pneumonia.
   - TREATMENT.—Cut away all the affected parts.
     - Rub pure carbolic or nitric acid into raw surfaces.
     - Antistreptococcal serum.
     - Subsequent plastic operation.
2. ACUTE SPREADING GANGRENE, or Malignant œdema, or Acute emphysematous gangrene, or Acute traumatic gangrene.
   - Predisposing causes: Chronic alcoholism—Diabetes.
   - Exciting cause: Large lacerated wound with much dirt contamination, or virulent infection of a small wound.
   - ORGANISMS. (*a*) Bacillus of malignant œdema.
     - (*b*) Bacillus aerogenes capsulatus.
     - (*c*) Bacillus œdematis aerobius.
     - Usually mixed with other bacteria.
     - (*a*) and (*b*) are anaerobic; (*c*) is aerobic.
     - All have intense peptonizing action and produce gas by their growth. They are commonly found in garden soil.
   - SIGNS.—Occur within 24 hours of infection.
     - Rapidly spreading acute cellulitis—Parts become dark coloured—Surgical emphysema is well marked—Sloughing only occurs if the patient lives long enough.
   - SYMPTOMS.—Acute septicæmia—High temperature, later becoming subnormal—Delirium succeeded by coma.
   - TREATMENT.—Immediate high amputation.

Treatment of Gangrene.

I.—RENDER THE PARTS AS ASEPTIC as possible.

Spirit and biniodide of mercury 1-500 are specially valuable, because the spirit drys and hardens the skin.

II.—KEEP THE PARTS DRY and encourage evaporation.

This maintains a dry type of gangrene, or
Converts a moist into a dry gangrene.
Dry sterilized dressings applied with all aseptic care.
HOT FOMENTATIONS should be used with great caution:
- Never in cases of dry or aseptic gangrene.
- But only when sepsis is obviously present in moist gangrene.
- A fomentation is the surest way of converting a dry aseptic into a moist septic gangrene.

Treatment of Gangrene, *continued.*

III.—ELEVATION OF THE PARTS, KEEPING THEM WARM.

To encourage the circulation.

To empty the veins, and thus prevent the moist type of gangrene.

To prevent the spread of the gangrene in cases where the collateral circulation is very feeble.

IV.—AMPUTATION.—In all cases except, perhaps,

(*a*) Senile gangrene with marked calcareous arteries and feeble general condition or albuminuria.

(*b*) Diabetic gangrene with marked acetonuria (acidosis). or commencing coma.

(*c*) Acute infective gangrene which has involved the trunk, or in which septicæmia has already developed.

WHEN AND WHERE TO AMPUTATE must be determined by

(*a*) The nature of the gangrene, dry or moist.

(*b*) The presence and virulence of septic processes.

(*c*) The condition of the main blood-vessels.

(*d*) The general condition of the patient.

WHEN TO AMPUTATE (i.e., at what period) ?

In dry gangrene: Directly a definite line of demarcation appears, or directly the final death of the part is inevitable—When pain and exhaustion are intolerable.

In moist gangrene: Directly active septic processes or a line of ulceration are evident.

WHERE TO AMPUTATE ?

With diseased vessels: High amputation, e.g., lower third of thigh for senile gangrene of toes.

With active sepsis but healthy vessels: Amputation well above the inflamed area, e.g., middle of calf for gangrene of foot.

With healthy vessels and no sepsis: Amputation through the line of demarcation, or just above it.

FROST-BITES AND BURNS.

Frost-Bites.

CAUSES. (1) Direct effect of cold on exposed parts; (2) Pressure of inflammatory exudation caused by too rapid thawing.

CHARACTERS.

(*a*) SUPERFICIAL DRY GANGRENE of nose, ears, fingers, or toes—The slough which separates is superficial—The process is painless—It occurs as the direct effect of the cold.

(*b*) MOIST GANGRENE of an inflammatory character from too rapid thawing—Is extremely painful—Often ends in superficial ulceration rather than actual gangrene.

TREATMENT.—Very gentle gradual thawing—Rubbing with snow-water—Elevation of part with gentle bandaging to prevent congestion—Keep part aseptic, dry, and elevated when gangrene has occurred.

Burns and Scalds.—Produce inflammation, ulceration, or gangrene.

DEGREES OF INJURY produced by burns or scalds.

- 1st Degree.—Erythema—Local congestion of the skin. Only dangerous if extensive, and then shock and widespread superficial vasomotor paralysis may cause death.
- 2nd Degree.—Vesication—Epidermis is raised in blisters—Danger to life is the same as in 1st degree.
- 3rd Degree.—Destruction of epidermis—Raw dermis with naked nerve papillæ is exposed—The most painful form of burn. Healing is rapid, regeneration complete—No contraction results.
- 4th Degree.—Destruction of the dermis—The nerve terminals are destroyed—All true skin structures are lost in affected area. Healing involves great and deforming contraction—Scar formed is merely fibrous tissue covered by epidermis, and contains no proper skin elements, hence is devoid of hair, sweat and sebaceous glands, and but few nerves, lymphatics, or blood-vessels.
- 5th Degree.—Destruction below the layer of deep fascia—Muscles are injured or destroyed—Deep muscular fibrosis results from healing.
- 6th Degree.—Charring to the bone or total gangrene.

  The last three degrees involve all the dangers of inflammatory fever and septic absorption.

STAGES IN THE COURSE OF A BURN.

- 1st Stage. Shock.—At the time of injury: Severe (*a*) in proportion to the extent of the surface; (*b*) In children; (*c*) In burns of trunk, especially abdomen.

  When the shock is fatal, the internal organs, and especially the portal system, are found engorged, as though from vasomotor paralysis.
- 2nd Stage. Inflammation.—During the separation of dead tissues. Lasts one or two weeks, in proportion to the depth, extent, and septic infection of the burn. Any variety of septic complication may ensue—Gastro-intestinal inflammation, ulceration may result from septic absorption—Duodenal ulcer in an acute form may cause death from perforation or bleeding.
- 3rd Stage. Healing.—From time of separation of dead tissues until recovery—Will vary according to the degree—In first 3 degrees merely involves vascular changes or regeneration of epidermis from dermis—In 4th and 5th degrees will involve the cicatrization and contraction of an ulcer—In 6th degree it will be similar to the changes which follow massive gangrene.

CAUSES OF DEATH.—Shock—Gastro-intestinal inflammation and ulceration—Septic absorption.

TREATMENT.

- In First Degree.—Dust with boracic acid, and cover from the air.
- In Deeper Burns.—Render aseptic (under anæsthetic if necessary).

Burns and Scalds, *continued.*

Dress with oiled lint, carron and eucalyptus oil (10 to 1), or picric acid.

Graft skin if the wound is large, and if the whole thickness of skin is gone.

Amputate only if a limb is clearly rendered useless, or if severe sepsis occurs.

SKIN-GRAFTING.

Uses.—(1) To facilitate healing of any large superficial wound when much skin is destroyed, or (2) Any large ulcer with great loss of skin; (3) When as the result of a burn of the 4th or worse degree a wound is left which can only heal by great contraction.

Methods.

1. REVERDIN'S.—Minute fragments of epidermis only. Hardly ever used now.
2. THIERSCH'S.—Large pieces of epidermis with some dermis.

   Is used when large areas have to be covered, e.g., after amputation of the breast, in closing a large burn.

   PREPARATION OF THE WOUND :—It must have no active sepsis or irritating discharges—It must be recent and vascular—Exuberant granulations must be scraped away, but small recent vascular granulations may be left—Bleeding must be stopped by gauze pressure over protective while grafts are cut.

   PREPARATION OF GRAFTS :—Skin must be rendered aseptic—Cut grafts with flat, sharp razor—Hold skin flat and tense with strips of wood—Keep razor surface wet with water all the time—Cut the grafts as large as possible—Best taken from the thigh.

   The cut should be down into the fibrous vascular layer of the dermis, but not through it.

   The graft beds are dressed with simple ointment, and heal quickly, because the deep layers of skin regenerate those taken away.

   LAYING THE GRAFTS :—Grafts have been placed in warm saline fluid—Laid on the wound absolutely flat—Wound surface should be completely covered—Dressed over perforated tin foil—Or left with no dressing in actual contact—Change the dressing about every three days—Use no chemical antiseptics throughout.
3. WOLFE'S.—Whole thickness of the skin. In covering small wounds, e.g., eyelids, or any part of the face, e.g., after the removal of rodent ulcers.

   Thin, hairless skin should be chosen, e.g., prepuce—parts of the outside of the arm.

   The graft is cut a little larger than required—Is sewn accurately to the edges of the gap—Firm pressure is needful afterwards.

   This method is now frequently used for the same class of case as Thiersch's grafts, and in much the same way, except that the grafts consist of the whole skin thickness from which the subcutaneous fat has been cut off.

CHAPTER V.

ACUTE SURGICAL INFECTIONS.

CELLULITIS.

Definition.—Septic inflammation of subcutaneous and fascial connective tissue.

Causes.

Injury, or wound, or abrasion of skin, or mucous membrane—especially deep wounds with small external openings.

BACTERIA: Staphylococci, (albus and aureus) (slight cases), streptococci (severe cases), or bacillus of malignant œdema (emphysematous gangrene).

PREDISPOSING CAUSES: albuminuria, diabetes, debility.

Symptoms.

INCUBATION PERIOD of about two or three days, during which there is some general malaise.

ONSET: Usually with a sharp rise of temperature and rigor—Rarely a subnormal temperature occurs, and this in the worst cases.

CONSTITUTIONALLY: All symptoms of fever, with great pain and sleeplessness—Delirium in very bad cases. There may be large subcutaneous abscesses (staphylococcus), or a spreading sloughing of the cellular tissue (streptococcus). The tendency is to spread up the limb along the lines of the fascial planes much more quickly than to burst through the skin.

LOCALLY all the signs of inflammation occur: Tenderness, redness, brawny induration and œdema—Intense throbbing pain—The outline and margin are very indefinite and ill-defined.

COMPLICATIONS.—Some form of serositis by extension, e.g., Meningitis from disease in the scalp or orbit—Pericarditis, Pleurisy, Mediastinitis, from cellulitis of the neck—Peritonitis from pelvic cellulitis—Septicæmia or Pyæmia from general infection.

Treatment.

MULTIPLE INCISIONS down to the deep fascia.

HOT FOMENTATIONS or immersion in continuous hot bath.

BIER'S PASSIVE HYPERÆMIA.

ANTISTREPTOCOCCUS SERUM in suitable cases.

Special Forms.

SCALP.—The affected parts are bounded by the occipito-frontalis aponeurosis, i.e., by the superciliary ridges in front, superior curved lines of the occiput behind, the temporal ridges at the side.

The whole scalp over this area may become lifted up by a bag of pus. MENINGITIS or necrosis of the skull may follow.

ORBIT.—Caused by penetrating wounds or extension from boils on the eyelids.

RAPID COURSE.—Very great pain, with high temperature and delirium. The eyeball is pushed forwards and vision lost.

COMPLICATIONS.—Meningitis, septic thrombosis of the cavernous sinus, suppurative panophthalmitis, optic neuritis, destruction of the ocular muscles and retraction of eyelids.

TREATMENT.—Free incisions and fomentations. The eyeball must be sacrificed by incision or excision in severe cases or where panophthalmitis supervenes.

CERVICAL OR SUBMAXILLARY CELLULITIS (LUDWIG'S ANGINA).—Affects the deep cervical fascia, usually beginning in the submaxillary region and spreading rapidly downwards and inwards.

CAUSED BY an intrabuccal or pharyngeal infection, usually in old and debilitated people.

RAPID SLOUGHING and suppuration occur in the plane of the deep fascia, at the root of the tongue, and spreading down the pretracheal fascia to the superior mediastinum and pericardium, or down the prevertebral fascia to the posterior mediastinum and pleura.

COMPLICATIONS.—Œdema glottidis—Sublingual abscess—Suppurative mediastinitis—Pericarditis—Empyema.

TREATMENT.—Free incisions, especially in the mid line above the hyoid bone—Tracheotomy for œdema glottidis.

CELLULITIS OF THE DIGITS—WHITLOW. (This may be less or more than a mere cellulitis, but in the main it is this, and therefore it is most conveniently described here).

CAUSES.—Small septic wounds of the fingers or toes, often under the nail.

VARIETIES :—

1. SUBCUTICULAR. Between the epidermis and dermis. A purulent blister is formed.
2. SUBCUTANEOUS. Rapidly spreadng cellulitis, which extends into the palm and on the back of the hand, and thence to the arm and axilla.
3. THECAL. A purulent synovitis of the flexor sheaths. Spreads rapidly up these. In the case of the little finger and thumb the sheath is continued into the palm. In the three middle fingers it stops short at the head of the metacarpals. It is almost always combined with cellulitis, so that it is seldom possible to distinguish it from the latter. It often causes

sloughing of the tendons, and almost always leads to adhesions of the tendons on recovery.

4. SUBPERIOSTEAL.—A periostitis of one or more of the phalanges, usually the terminal, infected from the nail matrix. It causes a more limited inflammatory swelling than the other varieties, and often results in necrosis of the phalanx.

TREATMENT.—Incisions and fomentations.

Bier's hyperæmia has given the best results. When it is employed: (1) The incisions need not be so many or so free; (2) Drainage need not be used; (3) Recovery is much more rapid; (4) Sloughing is usually prevented; (5) Adhesions are prevented.

PELVIC CELLULITIS.

CAUSES.—Septic conditions of the uterus, lacerated cervix, punctured wounds of the rectum, diseases of the bladder and prostate.

SIGNS.—Constitutional signs are severe. The uterus is pushed to one side, and a phlegmon arises on one side of the pelvis and spreads up the round ligament or cord to the inguinal region. An abscess forms either in the pelvis or inguinal region.

DIAGNOSIS.—An inguinal abscess of this kind has to be distinguished from (1) Bubo, or other lymphatic swellings; (2) Inflamed hernia; (3) Psoas abscess; (4) Appendicitis.

COMPLICATIONS.—Abscess may burst into vagina, rectum, bladder, or intestine.

TREATMENT.—Hot douches and fomentations. Open through the vagina or the inguinal region.

ERYSIPELAS.

Definition.—A very contagious disease; consisting in an infective inflammation of the skin or mucous membrane.

Causes.

Predisposing: (1) A wound or abrasion; (2) Constitutional debility; (3) Bad hygiene.

Exciting: *Streptococcus erysipelatis*, which is indistinguishable from the *S. pyogenes*.

Pathology.—The lymphatics of the skin in the margin are crowded with chains of streptococci. In the regions where the disease has come and gone there is marked leucocytosis but no cocci.

Symptoms.—Malaise with a rigor and headache.

RASH appears within twenty-four hours. It appears first round the wound, which breaks open. It is of a vivid red colour, which fades on pressure. Pain and swelling are not much marked. The eyelids and scrotum when affected become very œdematous. Vesicles and bullæ form superficially, and a fine desquamation occurs, with some staining of the skin as the rash fades away.

Erysipelas:—Symptoms, *continued.*

THE MARGIN is well marked, rather gyrate, slightly swollen, rapidly advancing.

Sloughing of the skin rarely occurs, and then usually in cases of scrotal affection.

LYMPHATIC GLANDS in the neighbourhood are enlarged and tender.

EXTENSION may occur by the lymphatics or veins to the deep structures, or pyæmia may be set up.

CONSTITUTIONALLY. Patient is very ill, with high temperature —102°–104°. Delirium is frequent, especially when the scalp is affected. Vomiting is common.

COURSE. Tends to spontaneous recovery in one to three weeks.

Varieties.

FACIAL ERYSIPELAS is often apparently idiopathic and recurrent. It is accompanied by great œdema. It is liable to be complicated by meningitis.

FAUCIAL ERYSIPELAS. Spreads from the exterior to the pharynx. Causes great swelling of the parts, with a tendency to œdema glottidis. Sloughing or ulceration may follow. Massive enlargement of the glands at the angle of the jaw.

SCROTAL ERYSIPELAS. Causes great œdema, and in children a tendency to sloughing.

CELLULO-CUTANEOUS ERYSIPELAS partakes of the character of both cellulitis and erysipelas, affecting the skin and subcutaneous tissue. The margin is less sharply defined; the tendency to general septic infection and sloughing of the skin is greater than in either of the simpler diseases.

Diagnosis.

THE EXANTHEMATA, especially scarlet fever. In these the rash appears at the same time in different parts of the body. The rash has specific characters, and there are other symptoms, e.g., coryza or tonsillitis.

CELLULITIS.—There is more swelling and œdema, and the margin is not well defined.

DIFFUSE ERYTHEMA NODOSUM.—Occurs generally on the legs, with few or slight febrile symptoms. Isolated and ill-defined nodules are its chief feature.

ERYTHEMA SOLARE (sun-burn) is limited to the parts exposed; it does not spread, and there is but little fever.

ACUTE ECZEMA.—There is a copious exudation, and little tendency to pass away.

Effect on other Lesions.—Chronic ulcers, simple, tuberculous, or specific, may rapidly heal. Sarcomata have disappeared after erysipelas, giving the basis for Coley's treatment of the former.

Treatment.

STRICT ISOLATION, especially from surgical patients.

FOMENTATIONS and dressings to relieve pain and limit the diffusion of the virus.

Painting with 40 per cent aqueous solution of ICHTHYOL after scarification.

Painting the healthy skin beyond the rash with irritants, e.g., IODINE or SILVER NITRATE, or by scarification to produce leucocytosis.

ANTISTREPTOCOCCUS SERUM, 20–30 cc., given subcutaneously twice a day in bad cases.

SEPTIC INFECTION.

Causation.—An infection by the ordinary pyogenic bacteria, which are derived from the skin or mucous membrane of the patient, or by contact with infected materials or instruments.

Local Signs.—The wound becomes inflamed, i.e., painful, with red and swollen edges. Cellulitis may spread to neighbouring structures. When a stitch is the infective agent, the signs may be later and less manifest, and appear round the stitchhole.

Local Treatment.—Removal of stitches, with hot fomentations. Drainage or packing with iodoform. Daily irrigation with peroxide solutions.

Constitutional Signs of Infection.—These may be of five different grades, according to the amount and character of the toxic agent: (1) SEPTIC TRAUMATIC FEVER; (2) HECTIC; (3) SAPRÆMIA; (4) SEPTICÆMIA; (5) PYÆMIA.

Septic Traumatic Fever is the fever which accompanies an ordinary septic wound. It is most marked when the wound is closed. The temperature rises to 101°–103°, often with a rigor. It falls rapidly when the wound is opened, and is at an end when the layer of granulations prevent further absorption.

Hectic Fever is caused by the CONSTANT ABSORPTION OF SMALL DOSES OF TOXINS. Usually the temperature rises to 99°–101° every evening. It accompanies chronic suppuration, especially of septic tuberculous foci.

Sapræmia is caused by the absorption of a LARGE DOSE OF TOXIN. A large or virulent infective focus exists (e.g., a septic placental remnant in utero, or a localized appendicitis), and from this toxins (but not bacteria) enter the circulation.

SYMPTOMS.

A RIGOR, with rapid rise of temperature to about 104°, is the first symptom. In the worst cases the temperature remains subnormal.

VOMITING AND DIARRHŒA, delirium or coma, dyspnœa, or albuminuria, may occur, according to the organs which react most to the toxins.

Sapræmia, *continued.*

The Usual Signs of Severe Fever—dry tongue, quick pulse, scanty urine, and headache—are the rule.

All the symptoms subside rapidly when the infective focus is freely opened and cleared out.

Treatment must be directed to the cause. Also hypodermic, venous, or rectal saline transfusions are of the utmost value in diluting and excreting the toxines.

Septicæmia is caused by the absorption and development of bacteria in the blood of the patient.

BACTERIOLOGY.

Streptococcus pyogenes is the cause in more than half the cases, and nearly all cases of puerperal origin, and most of those of ulcerative endocarditis.

Pneumococcus and *Staphylococcus* are next in frequency. *Bacillus coli*, *B. œdematis maligni*, and *Gonococcus* are rarer causative agents.

PREDISPOSING CAUSES.

Special virulence of the bacteria, e.g., in small post-mortem wounds—Special debility of the patient—Large lacerated wound areas.

SYMPTOMS.

Local Inflammatory signs may be of any degree.

Temperature rises to 104°–105° with a rigor, and remains fairly high without marked intermissions. The temperature becomes subnormal, whilst the pulse rises in fatal cases.

The symptoms are the same as those of sapræmia, but of a further intensity.

Blood-stained diarrhœa, blood and albumen in the urine, petechiæ in the skin, and rapid coma are all very significant.

COURSE.—It is usually fatal in five to seven days.

POST-MORTEM SIGNS.

These are mostly due to early decomposition and to Hæmolysis, i.e., solution of the red corpuscles. Coagulation and rigor mortis are ill marked. The blood serum is lakey. The intima of the vessels and heart are blood-stained. The serous cavities contain blood-stained exudation. The viscera, especially the spleen and lungs, are soft, pulpy, and congested, and their epithelium has undergone Cloudy Swelling.

Similar phenomena are seen after death from all acute infectious diseases, also in sapræmia, pyæmia.

DIAGNOSIS.

In Sapræmia and Septic Traumatic Fever there are three points to help in the distinction: (1) The presence of an inflamed wound or local infective focus, in which the local signs are in proportion to the constitutional; (2) The rapid amelioration of symptoms on freely opening the infective focus; (3) The fact that repeated blood examinations gives a negative bacterial result.

HYPER-ACUTE EXANTHEMATA, in which the patient dies before the rash appears, are practically cases of acute septicæmia, indistinguishable from those of traumatic origin.

PYÆMIA is distinguished by the repeated rigors, the intermittent temperature, and local abscesses.

TREATMENT.

Local treatment on general lines must be undertaken, but is usually too late to avail.

Antistreptococcus serum, in doses of 30 cc. twice a day, is the best remedy.

Intravenous or other means of copious saline infusions help to eliminate the poisons.

Pyæmia.—Is caused by the diffusion of septic emboli throughout the circulation.

BACTERIOLOGY.

STREPTOCOCCUS is the commonest cause. *Staphylococcus, Pneumococcus, B. typhosus* are all rare.

CAUSES.—Infective phlebitis—Osteomyelitis—Infective endocarditis—Middle-ear disease—Puerperal infections—Any septic focus. In all these there is a septic thrombus formed in the veins, particles of which become washed into the general circulation.

PATHOLOGY.—Thrombi containing living bacteria form emboli which are caught in the capillaries of different organs. The lungs suffer first, and then the kidneys, brain, liver, spleen, and large joints. In portal pyæmia the liver suffers first and most.

Infarctions occur as wedge-shaped hæmorrhagic areas at each point of infection.

Abscesses develop from each septic embolus.

Some degree of septicæmia usually co-exists.

VARIETIES.

IN ACUTE PYÆMIA there is also marked septicæmia, and death occurs before any of the abscesses have time to become prominent.

IN CHRONIC PYÆMIA no bacteria can be found in the blood. The abscesses develop at intervals and attain a large size. They are often in bones or joints.

SYMPTOMS.—All those of septicæmia and sapræmia may be manifest.

RIGORS of extreme severity and regular recurrence are the distinguishing feature. Dyspnœa, cyanosis, and sweating follow each rigor. They occur at intervals of one or two days.

AN INTERMITTENT FEVER, with rises to 104°–106°, accompanying the rigors, is also well marked.

LOCAL INFARCTIONS AND ABSCESSES appear about the end of the first week. The visceral infarctions are usually small, but may be notified by sudden stabbing pain or the development of pleurisy.

Pyæmia, *continued.*

Superficial Abscesses, e.g., in joints, often develop rapidly and without any signs of inflammation. When opened there is no attempt at granulation or inflammatory repair.

DURATION.—Varies from ten days to several months.

POST-MORTEM SIGNS are those of septicæmia, together with: (1) Some primary focus, usually of a bony nature, in which necrosed bone is found with neighbouring veins filled by breaking down septic clots; (2) The infarctions or abscesses in different organs.

PROGNOSIS depends upon:—(1) The accessibility of the primary lesion; (2) The early treatment of the primary focus; (3) The vitality of the patient and the virulence of infection; (4) The presence of septicæmia as indicated by bacteria free in the blood-stream.

TREATMENT.

Local.—Radical treatment of primary focus—Opening and scraping septic wounds—Scraping out septic bony cavities—Amputation of a septic limb—Opening and plugging a septic sinus.

General.—Antistreptococcus serum—Tonic treatment by drugs and food.

TETANUS.

A toxæmia due to a local infection by the *Bacillus tetani.*

Causes.

PREDISPOSING.—Hot climates. Agricultural labourers and stable attendants are specially liable to contact with contaminated soil.

EXCITING.—A wound—generally septic, sometimes merely contused or punctured—is infected by the tetanus bacillus.

Bacillus.—Occurs in ordinary garden earth and in road sweepings. Consists of a delicate straight rod which develops a spore at one end (drum-stick bacillus). It is provided with flagellæ. It is a strict anaerobe, but can grow in surface wounds where pyogenic cocci absorb all the oxygen. It stains by Gram's method—Grows in the depth of stab cultures—Its spores are very resistant to heat and chemicals—It produces no effect on inoculation, unless the tissues are depressed by injury or toxic effect of sepsis.

Pathology.—The bacilli remain localized to the point of infection. The disease is a pure toxæmia produced by the absorption of toxins from the wound. The toxins travel to the central nervous system in the perineural sheaths. In the central nervous system the tetano-toxin acts something like strychnine as a powerful spasmodic poison.

Post Mortem: Redness and congestion of the nerves leading from the infected focus. Softening and ecchymosis in the brain and cord. Molecular changes in the motor nerve cells. Rupture of muscles.

Symptoms of Acute Tetanus.

INCUBATION PERIOD.—Three days to one month.

EARLY SIGNS.—Stiffness and cramps in the jaw muscles, and in the neck.

SPASMS.—Trismus or lockjaw—Rigidity of the cervical muscles—Facial spasm (risus sardonicus)—Pharyngeal spasm causing dysphagia—Trunk muscles producing opisthotonus (backward arching), emprosthotonus (forward arching), or pleurosthotonus (lateral arching)—Abdominal muscles, especially the recti, are strongly affected—Respiratory muscles are attacked last.

CHARACTER OF SPASMS.—Strong, very painful, and continuous (tonic) contractions, lasting several minutes at a time. Are determined by any sensory stimulus, e.g., light, sound, or a draught. Often cause rupture of the muscles.

TEMPERATURE is always high at the end (110°), and is usually raised throughout. Sweats are profuse.

CONSCIOUSNESS is unimpaired, and the expectation of the convulsions produces great mental suffering.

DEATH occurs in two to six days from exhaustion or asphyxia.

Chronic Tetanus is the milder form of disease. The incubation period is longer, fever is absent, and the convulsions much more limited and less severe.

Cephalo-Tetanus is a special form following some head injuries. Facial paralysis occurs in conjunction with other symptoms, e.g., spasms. Pharyngeal spasm and mania are prominent, and give it a resemblance to hydrophobia.

Diagnosis.

STRYCHNINE-POISONING causes similar symptoms, but of much more rapid onset. The muscles become quite relaxed between the spasms, and the hands are often affected.

HYDROPHOBIA.—The convulsions are clonic—hallucinations and mania well marked. The muscles of deglutition and respiration are markedly affected.

SIMPLE TRISMUS, as from dental caries or affections of the temporo-maxillary joint, shows no affection of the neck muscles, and is generally unilateral.

Prognosis.—Is always bad, but when the incubation period is long there is a better chance of recovery. Whereas 96 per cent die when the incubation period is under ten days, only 55 per cent die if it is three weeks or over.

Treatment.

THOROUGH CAUTERY or EXCISION of the primary lesion.

ANTITETANIC SERUM is a pure antitoxin, and has little effect on the toxins which have already combined with the cells of the central nervous system.

Treatment, *continued.*

Injected (1) Subcutaneously, or (2) Into the nerve sheaths, in doses of 30 cc., followed by 15 cc. twice daily; or (3) Intracerebral, by a small drill opening through the skull into the depth of the brain: 25 cc. of dried serum are used with 5 cc. water; (4) By means of a lumbar puncture.

FEEDING is by a rubber tube behind the teeth or through the nose.

Absolute QUIET and avoidance of all sensory stimuli.

DRUGS.—Chloral, bromide, and chloroform; hypodermic injection of ten minims of 2 per cent solution of carbolic acid three times a day.

ANTHRAX.

The Bacillus Anthracis is large and long: 5 to 20 μ long and 1 to 1·5 μ wide. It is aerobic, and liquefies gelatine. It forms spores when growing outside the body in the presence of oxygen. These spores are very resistant to chemicals and heat.

Infection usually takes place by the contact of butchers, graziers, leather or wool workers, with the diseased sheep or cattle. In the latter it causes splenic fever, and the beasts, their carcases, and skin retain the infection.

Malignant Pustule is the result of cutaneous inoculation by anthrax. Usually occurs on the face, neck, or arms. (1) An angry red pimple; (2) A crop of vesicles upon an infiltrated base; (3) A central black slough surrounded by vesicles and a large zone of œdema, are the stages in its appearance which it goes through in four or five days. Then the temperature rises and grave septicæmia is caused by the distribution of the bacilli. Special symptoms may be due to the involvement of the lungs, intestine, or nervous system.

Anthrax Œdema is a more virulent form of local infection, in which widespread œdema with multiple cutaneous sloughs precedes a rapidly fatal septicæmia.

Woolsorters' Disease is an anthrax septicæmia without external lesion. Usually the infection is by the lungs, and a severe PLEURO-PNEUMONIA is set up. More rarely the intestine is the primary focus, and painful diarrhœa with passage of blood occurs, as the symptom of ENTERITIS. In either case the result is usually fatal.

Treatment.—EXCISION of cutaneous foci of infection. Early injection with SCLAVO'S SERUM.

HYDROPHOBIA.

Cause.—Transmission of the virus by the bite or lick of a rabid animal, usually a dog.

Rabies in the Dog occurs three to five weeks after infection. It manifests three stages: (1) An altered disposition, in which the dog sulks or is snappy; (2) A stage of maniacal excitement, in which it attacks anyone it meets; (3) A paralyzed condition affecting the hind limbs and jaw. The dog throughout has no dread of water, but drinks freely.

Hydrophobia in Man.—Incubation period is six weeks up to a year.

A mental change—terror, delusions, and suspicions, with insomnia—is the first symptom group.

Clonic contractions of the tongue and pharynx, which later spread to other parts as general convulsions. Any sensory stimulus gives rise to these, especially the sight of water or any attempt to swallow.

Mouth is filled with tenacious mucus.

Respirations are irregular.

Violent homicidal mania is a rare occurrence.

Death occurs in two to seven days from exhaustion or glottic spasm.

Post-mortem changes are: Inflammation of the medulla oblongata, with engorgement of the salivary glands.

Treatment.—The dog should be kept under observation until the diagnosis is certain.

The bitten part should be EXCISED or CAUTERIZED after a tourniquet has been placed on the limb above.

PASTEUR'S TREATMENT should be tried as soon as possible. It consists in injections of preparations from the spinal cords of artificially inoculated rabbits, beginning with those whose toxicity has been rendered weak by long desiccation, and later using more virulent preparations.

When the disease has manifested itself, sedatives—chloral, bromide, and chloroform—are the only palliation.

Chapter VI.

GONORRHŒA.

Infection by Gonococcus.

CHARACTERS OF ORGANISM.—Stains easily—Gram negative—Occurs in pus or epithelial cells—Generally paired or in clumps—Capsule—Grows with difficulty on blood serum—Acids favour growth—Alkalies prevent it—It can penetrate intact mucous membrane—It prepares the way for ordinary pyogenic organisms.

SEAT OF INOCULATION.—Urethra (male or female)—Vagina—Rectum—Conjunctiva (especially in new born).

Symptoms of Gonorrhœal Urethritis appear 3–6 days after infection.

SIGNS.—Congestion of meatus—Glueing of lips of meatus together.

Discharge.—(1) Mucoid (stage of onset).
(2) Thick pus (acute stage).
(3) Thin pus } chronic stage.
(4) Gelatiform } chronic stage.

Glandular Enlargement occurs in severe complicated cases, especially when ulceration exists. It usually affects the inguinal glands.

SYMPTOMS.—Itching of meatus—Scalding micturition—Tenderness along course of urethra—Frequent micturition—Pain in perineum—Pain in back—Constipation—Some general malaise.

DURATION.—In favourable cases, one month; in chronic cases, several years.

Varieties of Gonorrhœal Urethritis.

I.—ANTERIOR URETHRITIS (all cases begin as anterior urethritis).—In front of compressor urethræ—Scalding micturition—First half of urine passed contains more shreds than second.

Complications. — Bubo — Balanitis — Chordee — Cowperitis — Stricture.

II.—POSTERIOR URETHRITIS.—(Occurs in the second week or later)—Behind compressor urethræ—Frequency of micturition—Pain after micturition—Pain in perineum—Second half of urine contains more shreds than first half—Many casts of prostatic ducts are in urine.

Complications.—Cystitis, prostatitis, epididymitis, and vesiculitis.

III.—GLEET.—Thin or gelatiniform discharge continuing for years after gonorrhœa.

Causes.—(*a*) Posterior urethritis where organisms are harboured in prostatic ducts; (*b*) Stricture of urethra; (*c*) Granular patches, i.e., ulcers of urethra, gout, alcoholism, venerism.

Diagnosis of Genital Gonorrhœa.

IN MALE.

Non-specific Urethritis.—Very rare—Organisms are Gram-positive—Clear up quickly.

Foreign Body in urethra (by sound).

Urethral Chancre.—(By induration.)

Chronic Purulent Cystitis or prostatic abscess (by passing catheter and examining rectum).

IN FEMALE.

Non-specific Vaginal Discharges are very common.

Leucorrhœa: Discharge comes from cervix uteri only.

Pelvic Abscess: Bimanual examination.

Carcinoma, Etc.: Bimanual examination.

Specific nature of discharge can only be determined by a demonstration of the gonococci.

Treatment of Gonorrhœal Urethritis.

IN ACUTE STAGE (frequent scalding micturition, free purulent discharge).

Rest. Copious fluid diet, e.g., milk, barley-water.

Forbid meat, alcohol, tea, coffee, and sexual intercourse.

Drugs.—Alkalies, e.g., potass. bicarb. gr. x—Potass. citras gr. xx.
Anti-Spasmodics, e.g., tr. hyoscyami ♏x.
Diuretics, e.g., infus. buchu ℥j.

IN LATER STAGE (scalding ceased, discharge less):—

Drugs.—Oleoresins: Ol. santali ♏x—Ol. copaibæ ♏x—cubebs, ʒj.

Urethral Injections: Zinc sulphate, silver nitrate, or protargol, gr. ij ad ℥j (strength may be gradually increased up to gr. v ad ℥j in each case, but pain should never be caused)—Potassii permanganas, 1-5000—Zinci-sulpho carbolas gr. v ad ℥j.

Directions.—Use after micturition. Inject 2–4 drachms. Unless posterior urethritis is already present, compress urethra against symphysis pubis when giving injection. Retain for three minutes.

Irrigation Treatment.

Use one or two pints of lotion. About quarter strength used for injection, e.g., potassii permanganas, 1–20,000; argenti nitras gr. x ad Oj; protargol gr. x ad Oj.

(*a*) For anterior urethra. Pass small catheter (No. 7) as far as compressor urethræ, and allow lotion to flow from an elevation of about one foot.

(*b*) For posterior urethra. Fit douche nozzle to meatus. Raise douche can about two feet. Fill bladder and allow micturition.

Treatment of Gonorrhœal Urethritis, *continued.*

TREATMENT OF GLEET.—Ascertain the cause if possible.

If POSTERIOR URETHRITIS exists: Irrigations as above—Passage of large Lister's sound occasionally.

If STRICTURE exists: Dilate.

If GRANULAR PATCHES, i.e., ulcers, exist: Touch with argenti nitras, gr. x ad ℥j, through urethroscope.

Complications of Gonorrhœa.

BY EXTENSION FROM ANTERIOR URETHRA.

BALANITIS AND BALANOPOSTHITIS.—If phimosis exists, slit up foreskin.

INGUINAL BUBO AND ABSCESS.—Open.

LACUNAR ABSCESS.—Penile fistula.—Open from outside as early as possible.

CHORDEE.—Treat by bromides and cold compresses.

WARTS on genitals, especially in women.—Touch with nitric acid, cauterize, or dust with calomel.

COWPERITIS.—Perineal abscess. Foment. Open when fluctuating.

RETENTION OF URINE (spasmodic and congestive).—Treat by hip-baths, sedatives, and, if necessary, soft catheter.

STRICTURE.—Generally a late sequela.

GRANULAR ULCERS.

BY EXTENSION FROM POSTERIOR URETHRA.—Cystitis Epididymitis—Prostatitis—Vesiculitis.

IN THE FEMALE.—Cystitis—Labial abscess—Vaginitis—Endometritis—Salpingitis—Ovaritis—Peritonitis, generally localized in pelvis, and causing adhesions and sterility, sometimes general and acute.

BY TRANSMISSION OF VIRUS.

PROCTITIS.—Tenesmus and discharge.—Treat by irrigations and astringents.

RHINITIS.—Alkaline irrigations.

CONJUNCTIVITIS.

In adult (unilateral).—Treat by Buller's shield over good eye—Irrigate with boracic acid—Instil argenti nitras, gr. v ad ℥j, 4tis horis.—Cover with iced compresses.

In infants (ophthalmia neonatorum—bilateral). Often causes corneal ulcers, corneal opacities, perforation of the eye, blindness.—Treat as above, cutting the external tarsal commisure if necessary for drainage.

Complications due to Absorption.

SEPTICÆMIA.—Very rare except as an arthritis.

"RHEUMATISM."—At least three forms occur late (3rd–4th week) in an acute attack, or more commonly late in chronic gleet.

1. SYNOVITIS WITH EFFUSION.—Generally one joint.
2. INFLAMMATION OF EXTRA-ARTICULAR FIBROUS STRUCTURES, tendons, tendon sheaths, and ligaments.—Causes first,

softening and stretching of ligaments, and subsequently, organization and adhesions. Especially seen in wrist, ankle, and sole of foot.

3. A TRUE ARTHRITIS.—Seropurulent effusion—Destruction of cartilages and ligaments—Permanent ankylosis—Affects large joints, especially knees, hips, wrists, ankles. Often two or three joints are simultaneously affected, and all clear up except one.

PATHOLOGY.—Due to absorption of toxins, gonococci, and other pyogenic organisms.

TREATMENT.—Cure the urethral condition. (This cures the majority of cases included under 1 and 2, showing that these are probably sapræmic.) Rest, pressure, and Scott's dressing in inflammatory stage, massage and passive movements later. In group 3, where subacute destructive arthritis exists, open joint and irrigate.

CHAPTER VII.

SYPHILIS.

Definition.—Infective venereal disease peculiar to human subjects, but inoculable into higher anthropoid apes.

Cause.—Infection by a specific spirillum, the *Spirochæta pallida*. Inoculation of any thin or abraded skin or mucous membrane.

Sources of Infection.

PRIMARY LESIONS.

SECONDARY LESIONS, especially condylomata.

INDIRECTLY, e.g., by pipes used by those with lesions of the mouth.

Inheritance.

BY SPERMATOZOON—Paternal transmission.

BY OVUM—Maternal transmission. The mother has syphilis before conception.

BY PLACENTAL CIRCULATION, i.e., mother conceives, then acquires syphilis, and then infects fœtus. This mode of transmission probably never occurs, because the placenta filters out the spirilla.

If healthy mother bears a syphilitic child (paternal transmission) she becomes immune from the disease (Colles's law).

Stages of Syphilis.

PRIMARY.—Local manifestation at the seat of inoculation. Absent in inherited syphilis.

SECONDARY.—General infection. Symptoms occur from one month to two years after inoculation. Lesions are superficial, symmetrical, and infective.

TERTIARY.—Lesions appear from second year onward. They consist of deep fibrocellular infiltrations of skin, bones, and viscera. They are not infective.

Incubation Period.—One month—2–6 weeks.

Primary Lesion.—Hard or Hunterian chancre.

POSITION in order of frequency.

PENIS.—Glans or inner surface of prepuce—Outside skin—Meatus (common)—Urethra (very rare).

VULVA, vagina, or cervix.

Lips, anus, fingers, tongue, breast, abdomen, palate, and tonsils.

CHARACTERS OF HARD CHANCRE.—Usually single—Appears 2–6 weeks after infection—Begins as a papule, which ulcerates later—Base is indurated like thin cartilage—Painless and without inflammation—Associated lymph glands become "shotty," i.e., enlarged, but remaining painless and discrete and freely movable—Lymph vessels between chancre and glands are often thickened and indurated.

It is followed by signs of specific syphilitic infection. This is the only certain proof of the nature of a hard chancre.

Heals spontaneously.

Leaves little or no scar.

DIAGNOSIS OF PRIMARY SYPHILIS.

Soft Chancre.—Generally multiple—Appears 3–6 days after infection—Consists of a deep ulcer, which rapidly extends, and has sloughy base—Copious foul discharge—All signs of inflammation—Lymph glands become inflamed and matted together and suppurate—No induration except inflammatory exudation—Will not heal without local treatment—Leaves well-marked scar—Is not followed by general infection.

Chancre of Mixed Infection.—Presents signs of soft chancre at first—Later becomes indurated, and is followed by signs of secondary syphilis.

Primary Syphilis and Gonorrhœa.—Inflammatory complications of gonorrhœa may mask hard chancre—Indurated sore, followed by secondary syphilis.

Concealed Chancre.—Phimosis may conceal sore and produce a good deal of discharge—Bullety bubo and later secondaries.

Epithelioma.—Patient generally over 50—Longer history—Foul discharge—Warty growth with hard base, or ulcer with everted hard edges.

Gumma or "Relapsing Chancre."—Associated with other tertiary lesions—Past history of primary and secondary syphilis—Inguinal glands not enlarged.

Psoriasis—Boil—Inflamed sebaceous glands—Eczema—Lupus.

COMPLICATION OF CHANCRE.—Phagedenic ulceration, especially when occurring under a tight prepuce.

TREATMENT OF PRIMARY SYPHILIS.

If chancre is Dry. No local treatment is necessary.

If chancre is Moist, Ulcerated, or Soft:

Lotio nigra or iodoform ʒj ad boracic acid ℥j.

If chancre is Foul and Rapidly Spreading:

Pure carbolic acid, or acid nitrate of mercury.

If chancre is seen within a few days of its appearance, and is situated on the Prepuce:

Excise by circumcision.

If chancre is concealed by Phimosis:

Slit open prepuce.

Never give constitutional treatment until diagnosis is certain, i.e., in most cases until secondaries appear.

Secondary Syphilis.

APPEARS one to two months after chancre ; two to three months after infection.

LASTS any time up to two years from infection.

GENERAL CHARACTERS OF SECONDARY LESIONS.—Lesions affect skin, mucous membranes, lymph glands, and eyes — Copper colour — Polymorphic — Roughly symmetrical — Tend to spontaneous recovery—Without pain or irritation (secondary ulcers and fissures in mouth are painful).

GENERAL SYMPTOMS, which appear just before eruptions.

ANÆMIA.

ACHING PAINS in bones and loins, worse at night.

GENERAL ENLARGEMENT OF THE LYMPHATIC GLANDS. Most noticeable in posterior triangle of the neck and internal epitrochlear region.

A FEBRILE ATTACK, occasionally lasting a few days.

SKIN ERUPTIONS OF SECONDARY SYPHILIS.

1. ROSEOLA OR ERYTHEMA :
   Chiefly over trunk and flexor surfaces of limbs.
   Patchy erythema fading on pressure.
2. PAPULAR :
   All over the body. Very characteristic round forehead : "corona veneris."
   (*a*) SQUAMOUS, OR PSORIASIS : When papules are covered by scales of desquamating epithelium.
   (*b*) CONDYLOMATA, or moist papules on the skin : Papules present in surfaces of skin which are kept habitually moist. Occur in order of frequency : Round anus—Scrotum — Perineum — Vulva — Lips — Umbilicus — Axillæ — Under breasts — Between toes — External ear.
   These erythematous and papular eruptions are the only ones seen at all commonly. They are quite superficial, and leave no scar.
3. PUSTULES : Resembling acne—When a number of pustules are close together, and break, and become covered by a large scab, it is termed
   (*a*) Ecthyma.
4. TUBERCULAR : Deep layers of skin infiltrated—Ulcerate at one part and heal at another—Nose, forehead, scalp, buttocks.
5. BULLOUS : pemphigus—Large vesicles which suppurate, break, and are covered by a conical crust.
6. RUPIA : Deep ulcers covered by limpet-shaped scabs.
   Ecthyma, pemphigus, and rupia only occur late in very severe cases. Pemphigus is very characteristic of a severe form of congenital syphilis. They all leave scars which are characterized by being circular or gyrate in outline, thin, supple, and white.

AFFECTIONS OF THE HAIR AND NAILS.

ALOPECIA.

1. General shedding of the hair and eyebrows during secondary stage—Yields to general treatment.
2. Patches of hair follicles are destroyed by deep ulceration, either secondary, tubercular, or tertiary—Incurable.

ONYCHIA.—Nails may become grooved, brittle, and broken in late secondary syphilis.

PERIONYCHIA.—Secondary ulceration round nail margin—May cause nails to be shed.

AFFECTIONS OF THE MUCOUS MEMBRANES of the mouth and throat.

SIMPLE CONGESTION of faucial mucous membrane.

BALD AREAS on tongue.

MUCOUS PATCHES, i.e., papules on mucous surface.

FISSURES, especially near angle of lips and near edges of tongue.

ULCERS. Superficial, covered by grey membrane on tonsils and fauces. They spread by narrow grey margin—the "snail-track ulcers."

ŒDEMA GLOTTIDIS occasionally complicates ulcerations at the back of the mouth and pharynx.

STOMATITIS DUE TO MERCURY—Spongy bleeding gums—Profuse salivation—Fœtid breath—Ulcers with dark sloughy bases.

TREATMENT OF ORAL SECONDARY SYPHILIS.

Avoid alcohol and tobacco.
Cleanse teeth carefully.
Gargles of alum or chlorate of potash.
Touch ulcers with chromic acid, gr. x ad ℥j.

SECONDARY AFFECTIONS OF THE EYES.

IRITIS: Marked congestion, effusion, and adhesions—Pain slight.

CHOROIDITIS, RETINITIS (both rare).

LATE SECONDARY LESIONS—sometimes termed "reminders" —Sometimes forming intermediate or early tertiary signs.

Peeling patches on palms and soles—" syphilitic psoriasis."
Symmetrical painless epididymitis.
Synovitis, especially of knees.

Tertiary Syphilis.

Lesions occur generally from second year after infection onward to an indefinite period.

Are non-infective.

Do not cause secondary lymph-gland enlargement.

Asymmetrical.

Affect the skin and deep tissues, especially Fibrous tissues—Bones—Viscera.

Tend to (*a*) Deep ulceration; or (*b*) Formation of a gumma; or (*c*) Fibrosis, i.e., hypertrophy of connective tissue and atrophy of parenchymatous tissue of affected organs.

No tendency to spontaneous cure.

Are generally amenable to iodides.

Tertiary Syphilis, *continued.*

CHARACTERS OF A GUMMA.—Begins as collection of small round cells round blood-vessels in connective tissue. Blood-vessels are occluded by (*a*) Endarteritis obliterans; (*b*) Pressure of small-celled infiltration. Central mass of cells die and form central "gummy" core. Suppuration general supervenes in superficial gummata. When skin or mucous membrane have burst, a "wet wash-leather slough" is seen. Healing involves deep scarring with great contraction. Is generally painless and without signs of inflammation.

GUMMATOUS ULCER formed by bursting of superficial gummata. History of preceding lump. Deeply punched-out edges. Surrounding tissues healthy. Base formed by "wash-leather" slough. Granulations are firm, fibrous, and avascular. Outline is often formed by coalescing circles formed by fusion of small gummata. Painless. Scars left are thin and supple, with pale centre and pigmented margin.

TERTIARY LESIONS OF THE SKIN.

GUMMATA, probably of subcutaneous origin—Commonest in region of knees—Loins—Sacrum and buttocks.

LUPOID ULCERATION.—Tuberculous masses in the skin coalesce, ulcerate at one part and heal at another—Serpiginous outline—Especially attacks face, but may occur anywhere—Copper-red colour when active—Supple white scar when healed.

TERTIARY LESIONS OF THE BONES.

PERIOSTITIS: Local, plastic, forming "hard nodes"—Diffuse, combined with sclerosing osteitis, causing osteosclerosis.

GUMMATA: Subperiosteal, forming "soft nodes"—Central, causing expansion with possible spontaneous fracture.

NECROSIS OR CARIES: Probably resulting (*a*) from blood supply being cut off by gummata or osteosclerosis, or (*b*) by secondary septic processes.

SPECIAL BONES AFFECTED.

NASAL: Necrosis, perforation of septum, sunken bridge.

HARD PALATE: Perforation into nasal cavities.

CRANIAL VAULT: Especially frontal and parietal bones—One or more circular ulcers, or extensive "worm-eaten" surface, black colour, necrosing fragments—Great thickening from osteosclerosis.

TIBIÆ, clavicles, femora, forearm bones.

STERNUM.

PHALANGES: Especially proximal, causing syphilitic dactylitis—A local, hard periosteal thickening occurs in shaft of bone—It causes shortening of the bone, but seldom involves the joint—Painless, and rapidly heals under iodides.

To be diagnosed from tubercular dactylitis, which begins as central swelling at epiphyseal end of bone—Is very painful—Involves joints—Is not influenced by iodides.

LIPS, TONGUE, AND PHARYNX.

LEUCOPLAKIA.

FISSURES producing deep scarring and contraction.

GUMMATA. Commonest in dorsum of tongue—May destroy soft palate or produce extensive adhesions or pharyngeal stenosis.

LARYNX.

HYPERPLASIA of epiglottis or aryteno-epiglottidean folds.

GUMMATA and gummatous ulceration.

PERICHONDRITIS and NECROSIS of epiglottis or arytenoids, or rarely cricoid and thyroid cartilages.

Sometimes complicated by œdema glottidis.

RECTUM.

GUMMATA surrounding bowel, ulcerating into its cavity—Forming fistulæ—Producing commonest form of non-malignant stricture.

VISCERA.—In order of frequency: Testis, liver, spleen, heart, lungs, and any of the other viscera rarely.

Two forms: SOFTENING GUMMATA or FIBROUS THICKENING.

NERVOUS SYSTEM.

BRAIN: Gummata (usually begin in bones or meninges)—Obliteration of cerebral arteries—Aneurysm of cerebral arteries—Chronic meningitis—Paralysis of nerves, especially second, third, fourth, and sixth—General paralysis of the insane.

SPINAL CORD: Chronic meningitis—Subdural gummata—Locomotor ataxy.

Congenital Syphilis.—Often produces abortion or still-birth, caused by syphilitic disease of the placenta or visceral disease (especially fibrosis of liver, spleen, and lungs) of fœtus.

SYMPTOMS of congenital syphilis appear generally within three months of birth—Are those of secondary and tertiary disease, with following special features:—

EARLY RASH specially affects buttocks and genitals.

ULCERS round mouth, nares, and eyes leave radiating scars.

PEMPHIGUS is specially characteristic of a grave form. It may affect even the palms and soles.

GENERAL STOMATITIS, producing early loss of milk teeth and deformity of permanent.

PURULENT RHINITIS, producing "snuffles," loss of bridge to nose in later life, caries of spongy bones, with ozæna.

HIGH PALATE (due to disease or stunted growth of nasal septum).

CRANIUM: Craniotabes: Unossified spots in frontal or parietal bones. "Parrott's nodes" over frontal and parietal eminences, causing natiform or "hot-cross-bun" skull.

Both these lesions may result from, or be intensified by, severe rickets.

Congenital Syphilis, *continued.*

Long Bones, especially humerus, tibia, radius, and uln
Epiphysitis, producing thickening—Separation of epiphysis
"Pseudo-paralysis"—Suppuration in joints, the acu
arthritis of infants.

LATE SYMPTOMS, occurring especially during puberty a
adolescence:—

Gummatous and Lupoid ulceration, most usually round no
mouth, or knees.

Interstitial Keratitis occurs usually from 5 to 20—Bo
eyes affected, but one at a time—Cornea is infiltrated wi
round cells—Ground-glass opacity—Marked circumcorn
zone of congestion—Vascular patches on cornea ("salm
patches")—Tends to recover—Leaves white patches.

Deafness due to labyrinthine disease.

Sclerosing Osteoperiostitis or epiphysitis of long bon
May produce massive thickening, lengthening, or shortenin
These lesions are often symmetrical.

Affections of Teeth. In permanent set in order of frequenc
Central upper incisors, lateral upper incisors, lower inciso
first molars: show:—

Cutting edge or crown smaller than base, forming "pe
top" teeth.

Semilunar notch in cutting edge ("Hutchinson's" teeth

Treatment of Syphilis.

MERCURY.—To be given as pulv. hydrargyri c. creta, after mea
Dose, about gr. ij three times a day for an adult.

Combine with pulv. ipecac. co., if diarrhœa is caused.

Increase the dose until the gums become spongy as long as t
disease is advancing.

When all signs have disappeared give small dose (e.g., gr
t.d.s.) for Two Years.

Precautions.—Avoid mercurial poisoning, as seen by stomatit
diarrhœa, or tremors. If these occur, discontinue for a ti
and give in some other manner.

Keep teeth carefully brushed.

Gargle mouth with alum or chlorate of potash.

Other Methods of Administration.

In Mixtures, as liq. hydrarg. ʒj.

As an Inunction, ung. hydrarg. ʒij, rubbed into thin pa
of skin thrice weekly. For infants, ung. hydrarg. ʒj dilut
with two parts of ung. boracis, spread over inside of flan
binder.

By Vaporization. Calomel gr. xxx, vaporized over spirit lar
inside a blanket. Especially valuable when severe secondar
exist or when mercury by mouth causes stomatitis.

By Hypodermic Injection into gluteal region, using $\frac{1}{16}$ to $\frac{1}{10}$
hydrarg. perchlor., or metallic mercury in carboli
lanolin.

IODIDES.—Only necessary when tertiary symptoms are present.
Act by causing small cell infiltration to be absorbed.
Dose, gr. v. increased to gr. xxx t.d.s., or until action begins to be apparent. Give with ammonium carbonate, gr. v, and plenty of water.

RELATION OF MERCURY AND IODIDES.
Mercury cures the syphilis.
Iodides resolve the gummata produced by syphilis.
Therefore always give mercury when disease is progressing.
Combine it with iodides in tertiary stages.
Give iodides only, for solitary gummata.

FOR DEEP TERTIARY ULCERATION—Scraping or actual cautery combined with drugs is of great assistance.

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CHAPTER VIII.

TUBERCULOSIS AND ACTINOMYCOSIS.

TUBERCULOSIS.

Etiology.

PREDISPOSING CAUSES.

- INHERITED SUSCEPTIBILITY, especially flat or narrow chests—" Sanguine and phlegmatic dispositions."
- CHILDREN and young adults are specially susceptible.
- ATMOSPHERIC CONDITIONS. Bad ventilation, overcrowding, and dusty occupations are very potent causes.
- ANY LOCAL WEAKENING of the tissue resistance. Chronic catarrhal conditions—Strains, sprains, especially of bones and joints.

THE EXCITING CAUSE is the infection by the tubercle bacillus.

Tubercle Bacillus.—3 μ long and ·3 μ wide. Grows on blood serum or glycerinated agar-agar. Grows very slowly, first as small dots, then as wrinkled brown layer.

STAINS.—Carbol fuchsin, with heat, half an hour ; nitric acid, 1–4, few seconds ; alcohol, 60 per cent, half minute. Counter-stain with methylene blue.

Methods and Channels of Infection.—Through placental circulation (infinitely rare). Through respiratory organs. By breathing tubercle-laden dust. Through alimentary organs by eating tubercle-infected food. Through injuries and abrasions of the skin or mucous membranes.

Histology of a Tubercle.—Grey miliary tubercle is smallest tubercle visible to naked eye. It consists of a number of submiliary tubercles or giant-celled systems. Each giant-cell system consists of :—

- CENTRALLY-PLACED GIANT CELL.—Measures 50–500 μ. Many (20–50) nuclei massed near periphery. Bacilli are in the cell opposite to nuclei. Often presents coagulation necrosis (caseation). Found in other chronic granulation tissue.
- EPITHELIOID CELLS.—Placed in a zone round giant cells. Each is two to three times as large as a white cell. Elongated, with oval nucleus. Contain tubercle bacilli in or between them. Most characteristic feature of tubercle.
- LEUCOCYTES.—Form a peripheral zone. Contain no tubercle bacilli. Result from inflammatory reaction round tubercle.
- RETICULUM.—Fibrillar network lying between above cellular elements.

Later Stages in the tuberculous process.

CASEATION.—A number of miliary tubercles join to form a mass formed of all three kinds of cells. Blood vessels being scanty or absent, necrosis takes place. This cell necrosis produces a cheesy, structureless mass, known as caseous material. Caseation is best marked near the centre of the tubercle.

CICATRIZATION.—This is a process of repair. The granulation tissue surrounding the caseous centre is converted into fibrous tissue, and contracts. The liquid parts of the caseous material are absorbed, and the remainder forms a chalky mass—the so-called calcareous transformation.

LIQUEFACTION OR SUPPURATION.—This is similar to ordinary suppuration, but it takes place without the usual signs of inflammation. A chronic abscess is formed (*vide infra*).

PYOGENIC SUPPURATION.—In localities, e.g., the lung, when free access to the air occurs, or in any lesion where contamination from the skin or alimentary tract has taken place, a true pyogenic infection is set up which rapidly destroys the tissues and produces all the signs of pyogenic absorption.

Spread of Tuberculosis.

BY LOCAL EXTENSION.—The disease spreads by direct contiguity from one organ to another, e.g., from the lung to the pleura, or from a bone to the joint.

BY METASTATIC DEPOSITS.—Conveyed by blood, lymph, or secretions. Pulmonary tubercle often arises from that of the joints, tubercle of the bladder from that of the kidney, tubercular meningitis from tubercle of the testis.

BY GENERAL BLOOD DISSEMINATION.—A general miliary tuberculosis arises from the development of tubercles round all the small vessels, especially in the serous membranes.

Treatment.—As a general principle, this is a disease from which recovery will take place naturally, provided that the proper constitutional and local conditions be observed.

CONSTITUTIONAL CONDITIONS NECESSARY FOR RECOVERY.

FRESH AIR, especially dry, bracing, open air.

Abundant NITROGENOUS AND FAT FOOD.

LOCAL CONDITIONS NECESSARY FOR RECOVERY.

REST for the diseased part, both physical and functional. Hence the difficulty in treating tubercle in parts like the lung or bladder, which can never have complete physical or functional rest.

ABSENCE OF PYOGENIC INFECTION. When this infection has once occurred, natural cure is almost hopeless.

OPERATIVE TREATMENT is indicated generally when :—

The local focus is causing danger to life, e.g., tuberculous disease of the brain.

Treatment, *continued.*

The local focus is likely to infect other important organs, e.g., tubercle of one kidney or of one testis.

The local focus affects an unimportant organ, e.g., tuberculous glands of the neck.

Tubercular suppuration has occurred.

Pyogenic infection has occurred.

Other serious visceral disease is absent.

Rest and constitutional treatment have failed to cure.

RELATION TO OPSONINS.

INJECTIONS OF TUBERCULIN in doses of $\frac{1}{2000}$ - $\frac{1}{1000}$ mgram raise the natural powers of recovery by increasing the opsonins.

Useless when mixed pyogenic infections have occurred (e.g., in advanced pulmonary phthisis).

The injections should raise the opsonic index after a preliminary depression, and each injection should be given when the positive opsonic phase is at its maximum.

This raising of the opsonic index is useful as a preliminary to surgical operations.

No surgical operation should be done when the opsonic index is low. If it is, general tuberculosis is likely to follow.

Diagnosis.—In obscure cases the following methods are useful:—

INJECTIONS OF THE OLD TUBERCULIN.—Koch's tuberculin (·005 cc.) is injected hypodermically. A sharp reaction follows in a few hours, malaise, a temperature of 101°–104° F., with local pain and swelling of the affected focus. This is not free from danger, and is now seldom used except in the case of cattle.

INJECTIONS OF THE NEW TUBERCULIN (T.R.) in doses of $\frac{1}{1000}$ to $\frac{1}{2000}$ mgram. This affects the opsonic index in tuberculous cases. Thus: (1) A marked alteration in the opsonic index after tuberculin; (2) A variable opsonic index when taken on different occasions; (3) A considerable rise in the index after massage or exercise—all indicate tuberculosis.

CALMETTE'S OPHTHALMO-REACTION.—A drop of a watery preparation from the dried T.R. is instilled into the eye. Within six hours slight inflammation of the conjunctiva, especially at the inner canthus, indicates a positive diagnosis. It is not free from a slight risk of causing ulceration or severe conjunctivitis.

ACTINOMYCOSIS.

Etiology.—It is caused by infection by the *Streptothrix actinomyces* conveyed by infected corn or barley.

THE ACTINOMYCES grows in cultures in the form of long branching filaments. In the tissues it is arranged in the form of radiating club-shaped masses, from which it was named the ray fungus. These masses form granules in the pus which are visible to the naked eye.

IN CATTLE it commonly affects the tongue and jaw, forming chronic hard swellings, which break down to form suppurating, sinuses.

Distribution in Man.

The UPPER OR LOWER JAW, tongue, or floor of the mouth are attacked most often, and this by direct infection by diseased corn.

The LUNGS AND PLEURÆ are infected by inhaled particles, and chronic lesions like those of tubercle are caused. These may form an empyema, and the ribs and chest wall become the seat of chronic suppuration.

Any part of the alimentary tract, especially the CÆCUM, APPENDIX, and LIVER, are infected by swallowed particles.

Pathological Anatomy.—At first a hard, indolent nodule is formed, e.g., in the jaw. Then the soft parts are involved in the nodular mass, which then softens and breaks down, forming a multilocular abscess. This discharges pus containing the characteristic granules. The whole process is marked by pronounced fibrosis, producing a dense scarring, which, while it deforms the part, tends to limit the diffusion of the disease, which is therefore very chronic in its course. Secondary septic infection of the diseased areas occurs sooner or later, and is the ultimate cause of death.

Treatment is by local excision and erasion where possible, and by large doses of potassium iodide in other situations. As a rule the visceral disease is surely though slowly fatal.

CHAPTER IX.

TUMOURS.

Definition.—An abnormal overgrowth of tissue which subserves no physiological function.

CLINICAL CLASSIFICATION.

I. Innocent Tumours.

Encapsuled, or if diffuse they do not infiltrate.
Do not infect the lymph glands.
Do not recur after complete removal.
Do not disseminate.
Do not endanger life, unless they mechanically interfere with some vital organ, or unless they lead to hæmorrhage or sepsis.
Often multiple, and of different genera.

II. Malignant Tumours.

Are not encapsuled, but infiltrate surrounding tissues. Sarcomata are often in a capsule in their early history, but inevitably break through the capsule before long.
Infect adjacent lymph glands.
Tend to recur after removal.
Become disseminated in distant organs.
They inevitably destroy life.
Primary growth is always single, except for rare cases of bilateral sarcomata.

HISTOLOGICAL CLASSIFICATION.

I. Connective Tissue Tumours.

1.—LIPOMA (*Fat Tumour*).

SUBCUTANEOUS. May be sessile or pedunculated.
  Lobulated and encapsuled, and painless—the common kind.
  Multiple; painful—adiposus dolorosa.
  DIFFUSE—in neck, axilla, groin.
SUBSEROUS. In peritoneal cavity—In hernial sacs—Exaggerated appendical epiploicæ—In spermatic cord.
SUBMUCOUS. Rare—Small. Conjunctiva, larynx, and any part of alimentary canal.
SUBSYNOVIAL. Forming pedunculated masses protruding into joint cavities—" Lipoma arborescens."
INTERMUSCULAR.—Comparatively common.
INTRAMUSCULAR.—Rare.

PERIOSTEAL.—Mixed with striped muscle fibres, generally congenital.

MENINGEAL.—Either intra- or extra-dural—Often associated with spina bifida—Often congenital.

2.—CHONDROMA (*Cartilage Tumour*).—Often degenerate into mucus, or ossify—Long bones of limb at epiphyseal junction—Especially from phalanges and in rickets—Parotid, submaxillary, and lachrymal glands.

ECCHONDROSIS.—Local outgrowth of articular cartilages, especially knee.

LOOSE CARTILAGES.—One variety is formed by chondrification of synovial fringes, which then become detached and float in joint.

3.—OSTEOMA (*Bone Tumour*).

COMPACT OR IVORY.—Dense and sessile. From frontal bones, especially in sinus or orbit—External auditory meatus—Angle of mandible.

CANCELLOUS.—Tipped with cartilage until growth ceases. Generally from epiphyseal lines of long bones.

OTHER BONY OUTGROWTHS: Subungual exostosis, generally from great toe—Ossified tendon insertions—Ossified muscles.

4.—ODONTOMA.—(*Tumours developing from teeth or teeth germs*).

EPITHELIAL.—Probably malignant. Epithelial columns with cystic dilatation embedded in fibrous tissue. Arise from enamel organ.

FOLLICULAR.—A follicle containing a permanent tooth crown remains deep in the jaw and becomes distended like a cyst.

RADICULAR.—Hard mass of dentine and cement attached to root of tooth.

5.—FIBROMA (*Fibrous-tissue Tumour*).

SIMPLE.—Composed of masses of fibrous tissue. Gums (epulis)—Ovary—Uterus—Nerves.

MOLLUSCUM FIBROSUM.—Overgrowth of skin and subcutaneous tissue in pendulous folds or pedunculated tumours. Often associated with multiple neuromata.

6.—MYXOMA (*Mucoid Tumour*).—Very rare in pure form. Often present as degeneration product of other tumours. Nasal polyp (the common instance) is probably only œdematous granulation tissue.

7.—GLIOMA.—Consist of delicate, branching, neuroglial cells. Brain—Spinal cord.

8.—NEUROMA (*Nerve Tumour*).

TRAUMATIC.—Thickened proximal end of a divided nerve.

PLEXIFORM.—Myxomatous thickening of subcutaneous nerves underlying hairy mole.

"FALSE" neuromata are merely fibromata growing in nerve sheath.

9.—ANGIOMA (*Tumour consisting of Blood-vessels*).

SIMPLE NÆVUS.—Skin or subcutaneous tissue, mucous membrane.

Connective Tissue Tumours, Angioma, *continued.*

Birth mark, or port-wine stain, involves only capillaries.
Telangiectasis : Arterioles, venules, and capillaries.
Nævo-lipomata : Encapsuled masses of fat and nævoid tissues.

CAVERNOUS NÆVUS.—Skin, mucous membrane, surface of liver. Tissue similar to erectile tissue, containing large blood spaces.

PLEXIFORM ANGIOMA.—Subcutaneous tissue, especially in scalp or limbs.

Cirsoid aneurysm—Aneurysm by anastomosis, etc.

10.—LYMPHANGIOMA (*Tumours consisting of Lymph-vessels.*)

SIMPLE OR CAVERNOUS.—Dilated lymphatics. In skin, mucous membrane, especially tongue or lower lip.

LYMPHATIC CYST.—Cystic hygroma—Usually congenital—Neck, axilla, or groin.

11.—MYOMA.—Consists of unstriped muscle and connective tissue.

Occurs in uterus, bladder, œsophagus, and intestine.
Hard or soft, according to the proportion of fibrous tissue to muscle.
Generally multiple and encapsuled.
Tends to degeneration and other changes : Calcification—Myxomatous degeneration—Gangrene—Sarcomatous growth.

12.—MYELOMA or Myeloid Sarcoma (*Marrow Tumour*).

Consists of red marrow tissue, i.e., many multinuclear cells embedded in mass of round and spindle cells.
Very vascular, even pulsating.
Occurs in ends of long bones (especially head of tibia and lower end of radius), and in mandible or maxilla, or as an epulis.
Expands outer layer of bone to thin shell.
Does not disseminate or affect lymph-glands.
Does not recur if removed locally.

13.—SARCOMA (*Malignant Connective-tissue Tumour*).

Very vascular. May cause a bruit.
Liable to infect blood-stream by venous dissemination, and thus cause metastatic growths, most commonly in the lungs and liver.
Lymph vessels are absent or scarce. Many varieties never infect lymph glands. But sarcoma of the mamma and melano-sarcoma always infect the glands.
Degenerative processes, either hæmorrhagic or myxomatous, are common.
Consists of immature connective tissue.
Differs from innocent connective tissue tumour by preponderance of cellular over intercellular elements.
Differs from malignant epithelial tumour by fact that the cells are not arranged in groups, and that they are separated by intercellular tissue and blood vessels.

(*a*) ROUND-CELLED SARCOMA.—Universal distribution in tissues, and occur at any age.

Intercellular substance reduced to a minimum.

(*b*) Lymphosarcoma.—Tissue with structure of a lymph-gland, i.e., definite intercellular network enclosing small round cells.

(*c*) Spindle-celled Sarcoma (including the so-called "mixed celled sarcoma").—Cells are fusiform, or oatshaped. In sections some are round, others elongated.

Intercellular tissue often develops into fibrous tissue, cartilage, muscle, or bone.

Occurs in periosteum and in glands, e.g., ovary, testis, mamma, kidney, parotid.

(*d*) Alveolar Sarcoma. Cells arranged in alveoli like those of a carcinoma.

Originate in skin in hairy moles.

(*e*) Melano-Sarcoma. Contain masses of brown pigment between and in the cells—Cells vary in size and shape—Often of an alveolar type.

Originate in skin (moles), nail matrix, vulva, anus, palate, and uveal tract. At any age.

Intensely malignant. Infect lymph-glands and rapidly disseminate.

14. ENDOTHELIOMA is a sarcoma which arises from endothelial tissues, usually in a gland, e.g., the parotid or testis. It includes a quantity of glandular tissue, together with cartilage, and is very prone to myxomatous change. Also develops in the serous membranes—pleura and peritoneum—and from the "carotid gland" in the neck. The arrangement of the endothelium in branching columns gives a strong resemblance to an epithelioma.

Origin may be from endothelium:

Lining lymphatic clefts or spaces,
Lining blood- or lymph-vessels, or
Lining lymph-vessels in sheath of blood-vessels (perithelioma).

Psammoma is an endothelioma which arises from the endothelial cells covering the choroid plexuses of the brain.

Cells are arranged in whorls. Central portions become calcified.

15. ADRENAL TUMOURS.—Either sarcomata originating in the adrenal gland or in accessory adrenal glands, which may be situated beneath renal capsule, beneath hepatic capsule, or in the pelvis.

Imitate the structure of the adrenal zona fasciculata.

Often occur in infants, and are then generally bilateral.

II. Epithelial Tumours.

1. PAPILLOMA.—Consists of a central axis of vascular fibrous tissue surmounted by layer of epithelium. Epithelium never dips down into the tissue below the basement membrane.

Warts.—Occur on the skin, tongue, or larynx.

Single and stationary: May develop into horn, sarcoma, or epithelioma.

Epithelial Tumours, Warts, *continued.*

Multiple : Possibly infective—Especially in children—Often vanish rapidly.

Genital : Result from vaginal or urethral discharges.

VILLOUS PAPILLOMA. Delicate branching filaments occur in bladder or renal pelvis, and choroid plexuses of brain.

INTRACYSTIC PAPILLOMA. Occur in ducts of glands, e.g., mamma or ovary.

HORNS may arise in four ways :—

(*a*) By keratinization of a wart, especially on glans penis.
(*b*) By keratinization of the contents of a sebaceous cyst.
(*c*) By growth from the cicatrix of a burn.
(*d*) By overgrowth of a nail, especially the big toe nail.

2. EPITHELIOMA.—Consists in downward growing columns of stratified epithelial cells, simple or branching.

Begins as a wart, ulcer, or fissure.

Occurs in any surface covered by stratified epithelium—Mouth, tongue, œsophagus, larynx, anus, penis, scrotum, vulva, vagina, cervix—Skin, especially at site of warts, scars, or chronic ulcers—Bladder.

Columns of down-growing epithelium often become horny, forming cell nests or epithelial pearls.

Rapidly invade lymph-glands, but rarely become further disseminated.

3. ADENOMA.—Innocent tumour. Consists of gland tissue unconnected with ducts. Often encapsuled.

May occur in any gland. Common in mamma, ovary, parotid, thyroid, sebaceous glands, and uterus.

Alveoli having no duct outlets they are very often cystic.

When growing from a mucous surface they are pedunculated and called polypi. Common in rectum and uterus.

Usually occur in adolescence.

4. CARCINOMA.—Malignant tumour. Consists of mass of gland tissue of an abnormal type. Chief points are :—

Glandular alveoli penetrate below basement membrane.

Alveoli may consist of solid columns of cells instead of hollow tubes.

Irregular and disorderly proliferation of gland tissue.

Very liable to ulceration or necrosis.

Quickly infect lymph-glands—Rapidly disseminate.

Very rare in young—Commonest at 45–65.

(*a*) COLUMNAR-CELLED or acinous carcinoma arising from and mimicking a tubular gland structure.

Commonest in alimentary canal, œsophagus, stomach, colon and rectum, uterus.

(*b*) SPHEROIDAL-CELLED CARCINOMA. Solid columns of spheroidal cells.

Commonest in breast and stomach.

SCIRRHUS. A spheroidal-celled carcinoma, in which a large proportion of connective tissue occurs, forming a very hard growth. Especially common in old patients.

Encephaloid Cancer. A spheroidal-celled carcinoma, containing a minimum of connective tissue, forming very soft growths. Especially common in young patients.

(*c*) Rodent Ulcer or sebaceous carcinoma.

Probably starts in the cells of sebaceous glands—Begins as a nodule on the skin—Ulcerates after several years—Slowly destroys every tissue it meets, e.g., eyeball, bones of face.

Edges show but little heaping up—Often covered by a scab, but never heals by cicatrix.

Never infects lymph-glands. Never disseminates.

Commonly occurs on nose, eyelids, orbital angles, or cheek. May occur on neck, or rarely on trunk. Common in the aged, rare before 40.

Seldom recurs after complete removal.

Microscopically, it contains no "prickle" cells like an epithelioma, it never has cell nests or any keratinization, and its cells are smaller than those of an epithelioma.

5. CHORION EPITHELIOMA or deciduoma malignum.

Generally occurs in the uterus after a miscarriage.

Consists of tissue like that of the early fœtal chorionic tissue, viz. :—Multinucleated masses of protoplasm, the syncitium and many spindle cells like those of Langerhans' layer.

Exactly similar tumours occur in the testis; therefore they are, strictly speaking, teratomata.

III. **Dermoids.**—Skin or mucous membrane tumours occurring in situations normally devoid of these structures.

SEQUESTRATION DERMOIDS.—Occur in masses of embryonic epiblast which have become sequestrated in the deep tissues during development, especially along lines of fusion of epiblastic folds.

Situated. Mid line of body, except from tip of nose to occiput. In facial and branchial clefts, inner and outer canthus of eye, sides of nose, angles of mouth, pinna of ear.

Over scalp in the cranium and root of nose they are caused by patches of epithelium being cut off by inward growth of membranous bones from the surface skin. Hence these varieties either lie in a hollow of the bone or else underlie the bone altogether.

IMPLANTATION CYSTS.—Found under the skin or conjunctiva when pieces of epithelium have been buried by injury.

TUBULO-DERMOIDS.—Arise in a persistent fœtal duct.

Lingual.—Upper part of thyroglossal duct.

Thyroglossal.—Anywhere in mid line from hyoid to sternum, from persistence or diverticulum of thyroglossal duct.

Branchial.—Cysts beneath the deep fascia of neck arising from branchial clefts.

Rectal.—From post-anal gut. They may project into rectum as a polypus. They may lie between rectum and sacrum. They may bulge out behind as sacrococcygeal tumour.

Urachal.

Dermoids, *continued.*

OVARIAN DERMOIDS.—Probably arise from an ovum by an abnormal reproductive development, possibly by parthogenesis or by inclusion of a fertilized ovum within the body of the fœtus.

Contain skin and glandular structures of the utmost complexity: hair (often very long); glands (sebaceous, sweat, mammary); skin appendages (horns, nails, epithelial pearls); bone and teeth.

Somewhat similar tumours occur in testis.

MOLES.—Dermoid patches. Hairy, pigmented, raised areas of skin or conjunctiva.

Base consists of large cells arranged in an alveolar fashion.

Often form starting-place of melanotic sarcomata.

IV. **Teratoma.**—A conglomerate mass of fœtal tissues and organs.

Represents a "parasitic" fœtus, i.e., an incompletely separated and ill-developed twin.

Or possibly arises from activity of an undifferentiated reproductive cell.

Commonest in sacrococcygeal region.

Ovarian and testicular dermoids may be of this nature.

CYSTS.

Of Embryonic Origin.

DERMOID CYSTS (see above).

DENTAL CYSTS (see odontomes).

TUBULO-CYSTS. Dilatation of functionless ducts.

CYSTS OF THE URACHUS. Cysts of vitello-intestinal duct.

CYSTIC DISEASE OF TESTIS and cyst of the epididymis arise from Wolffian gland and duct.

PAR-OOPHORITIC CYSTS, PAROVARIAN CYSTS, and PARAVAGINAL CYSTS arise from Wolffian tubules and duct in female.

CONGENITAL AND INFANTILE HYDROCELES.

Distention Cysts. Formed by the distention of closed cavities.

GLANDULAR: Thyroid—Pituitary—Some ovarian.

NEW GROWTHS, especially adenomata: Adenoma of breast—Ovarian adenoma.

PERITONEAL: Hydrocele—Tunica vaginalis—Canal of Nuck—Funicular process—Ovarian—Hydrocele of Hernial sac.

SYNOVIAL.

BURSÆ.—Distention of normally-placed bursæ, e.g., prepatellar; bursæ connected with joints, so-called "Baker's" cysts; bursæ of new formation, e.g., that between hyoid bone and thyroid cartilage.

GANGLIA.—Hernia of synovial membrane through a tendon sheath.

VASCULAR SPACES or extravasation.

BLOOD CYSTS: Hæmatocele—Breaking down of vascular new growths—Arachnoid cyst.

LYMPH CYSTS: Hygroma—"Hydrocele" of neck—"Serous" cysts of mamma, axilla, or groin.

NEURAL CYSTS.

Hydrocephalus—Distention of ventricles—Syringomyelia—Distention of central canal of spinal cord—Meningocele, etc.—Spina bifida.

Retention Cysts.—Formed by a blocking of gland ducts.

Galactocele—Cystic disease of breast—Ranula—Dacryops—Pancreatic cyst—Hydrometra—Hydronephrosis—Hydrosalpinx—Hydrocholocyst.

Parasitic Cysts.

ECHINOCOCCUS.
CYSTICERCUS.

Hydatid Cysts.

DISTRIBUTION.—In Australia and the Arctic regions, where dogs are very intimately associated with human beings, the disease is common; elsewhere it is rare.

THE PARASITE is a small tapeworm, the tænia echinococcus, the adult form inhabiting the intestine of dogs and other animals, and the embryonic form growing in human tissues inside cysts.

LIFE HISTORY.—The adult consists of a worm half an inch long, with four segments. The head has four small suckers and a row of hooklets. The posterior segment is the largest and is filled with the genital organs. From these the ova are discharged, and are thence, by contamination with food, water, or uncooked vegetables, conveyed to the human host. The embryo develops in the stomach or intestine, and has four suckers and a set of hooklets. It burrows its way into the blood-stream, and is deposited in the liver or any other tissue. Here it forms a hydatid cyst.

THE HYDATID CYST consists of two layers: an ectocyst, which is firm and chitinous, and an endocyst, which is protoplasmic. The tissues of the host form a firm fibrous layer or capsule outside the cyst. The endocyst is formed by germinal protoplasm, from which generally grow: (1) Numerous daughter cysts; (2) Brood cysts containing other embryos in their interior; (3) Solitary heads of mature worms known as scolices. The fluid in the cyst is of specific gravity 1007, with a trace of albumen, some sodium chloride, and a quantity of free hooklets, by which the nature of the fluid is commonly recognized.

VARIATIONS.—If the cyst contains no brood cysts, daughter cysts or scolices, it is known as a barren or acephalocyst. Sometimes the main cyst disappears and a collection of daughter cysts and scolices lie free in the tissues. This is the common condition in hydatid disease of bones, and is known as exogenous development.

SITUATION OF CYSTS.—Liver, brain, kidneys, bone, or any other tissue, the first named being much the commonest.

RESULTS.

1. Simple growth, causing pressure symptoms.

Hydatid Cysts, *continued.*

2. Rupture into a serous cavity, causing toxæmia and marked urticaria. The scattered scolices may embed themselves and grow in the peritoneum or pleura.
3. Death of the parasite. The cyst then shrivels up, and may eventually become caseous or calcified.
4. Suppuration, with all the possible eventualities of an abscess, e.g., septic absorption, rupture, etc.

Cysticercus Cellulosæ is the intermediate or embryonic form of the common tape-worm or tænia solium. The cysticercus stage is generally found in the flesh of pigs, and the adult tænia in the gut of man. But sometimes, especially in Germany, the cysticercus develops in human tissues as one or many cysts of varying size. The commonest situation is the brain or eye, but occasionally it may form a cyst the size of a pea under the skin. In the brain they are eventually fatal. They cause destruction of the eye. From the skin they should be excised.

CHAPTER X.

WOUNDS.

Definition.—A forcible solution of continuity of the soft tissues.

Contusions.—Skin is not broken.

ECCHYMOSIS OR BRUISING produced by rupture of small blood-vessels.

If blood is poured out in some quantity, and enclosed by fascia or membranes, a

HÆMATOMA results. Fluid at first — Becomes hard when coagulation occurs—This hardness begins as a peripheral ring—Results in: absorption, organization in fibroid mass, serous cyst (e.g., arachnoid cyst), or suppuration.

TREATMENT.—Evaporating lotion, or hot fomentations. Firm pressure. Aseptic incision as a last resort in very exceptional cases.

Incised Wounds.—Clean cut, with little or no bruising. Wide gaping of lips. Free hæmorrhage.

TREATMENT.

1. ARREST HÆMORRHAGE.
2. RENDER PARTS AS ASEPTIC as possible.
3. SEW margins together.
4. DRAIN only if:—Deep infection cannot be got rid of—Deep bleeding cannot be arrested—Pressure cannot be applied (e.g., neck)—Wound communicates with deep cavity where extravasation exists, e.g., wounds of loin.
5. DRESS, with efficient application of
6. PRESSURE.
7. ATTEND TO THE GENERAL HEALTH.

FAILURE TO UNITE by first intention, due to:—

1. INEFFICIENT ARREST OF HÆMORRHAGE.
2. INEFFICIENT ASEPSIS, or foreign body in wound.
3. INEFFICIENT suturing.
4. ABSENCE OF DRAIN in cases mentioned.
5. INEFFICIENT DRESSING, i.e., infection after it has been sewn up.
6. GENERAL DISEASE.
7. CONSTITUTIONAL DEBILITY.

IF INFECTION HAS OCCURRED it is indicated by:—

Rise of temperature, rigor, etc.—Local pain and throbbing—Signs of inflammation round wound.

TREAT by: Opening freely—Washing out with peroxide of hydrogen—Freely draining.

DEATH MAY OCCUR from shock, hæmorrhage, or septic infection.

Lacerated and Contused Wounds.

Produced by a blunt instrument, which tears the soft parts.

Wound is associated with injury to the surrounding parts.

Hæmorrhage is slight, or absent, as vessels have been torn rather than cut.

Edges torn and bruised, with little or no gaping.

Parts may be cut off from their blood supply by—Original injury, inflammatory reaction (thrombosis), or pressure of bandages.

Hence SLOUGHING or GANGRENE are common occurrences.

IF ASEPTIC it may heal by first intention if edges can be brought together, but generally some sloughs have to be absorbed, or cast off, and healing is by granulation.

IF SEPTIC—as the majority of such cases are—acute inflammation, resulting in suppuration, sloughing, or gangrene, attack parts round the wound.

This secondary sloughing is responsible for the loss of much more tissue than is the original injury in most cases.

After sloughs have separated, wound heals by granulation.

DEATH MAY OCCUR from shock or septic infection.

TREATMENT.

Clean up (under anæsthetic if necessary). Trim off ragged edges and remove parts likely to slough.

Leave wound open, and pack or suture, leaving many gaps for drainage.

If septic inflammation occurs—Hot fomentations until sloughs have separated or suppuration ceased.

If wound is very severe and involves a limb :—

AMPUTATION WILL BE CALLED FOR :—If limb has been torn off—If part is quite disorganized—If gangrene occurs, or is imminent—If severe septic symptoms occur—In severe crushes of the foot in elderly patients.

AMPUTATION WILL BE NECESSARY in the following conditions if they result in GANGRENE, but not otherwise : Compound comminuted fractures—Extensive stripping of soft parts from bone—Wound of the main artery.

THE NECESSITY FOR AMPUTATION IS INCREASED BY :—Age and debility of patient—Distance from heart and bad blood supply (hence the foot is the most likely part to require amputation)—Severe sepsis.

Punctured Wounds.—Long deep wound with small orifice.

LIABILITY TO INJURE DEEP STRUCTURES :—Blood-vessels —Nerves—Viscera—Serous cavities—Joints.

If infected, deep suppuration occurs.

OFTEN LEAVES A FOREIGN BODY in depth of wound.

TREATMENT.

1. In the absence of evidence of sepsis or symptoms due to injury to deep structures—Simply clean and dress.

2. If deep structures give symptoms of injury—Open depths of wound, and deal with deep structures.
3. If there is any question of foreign body—Locate with X rays. Remove only if it is causing, or likely to cause, symptoms.
4. If suppuration occurs—Enlarge wound and drain.

Gunshot Wounds.

FROM MODERN RIFLE BULLETS.

Simple punctured wound.

Straight track between the aperture of entry—small round puncture,—and aperture of exit—a small slit.

Only injures structures in its direct path.

Sepsis, or introduction of foreign bodies, is rare.

SOFT-NOSED BULLETS, dum-dum bullets, and bullets of low velocity produce much more laceration from their disruptive force.

DEEP INJURIES.

BLOOD-VESSELS.—Fatal hæmorrhage—Traumatic aneurysm (when artery has been button-holed)—Arteriovenous aneurysm.

BONES.—Shaft: Compound comminuted fracture—Cancellous tissue may be simply perforated.

HEAD.—Splintering of the inner table—Destruction of cerebral substance along the track of bullet—Not necessarily fatal.

ABDOMEN.—Mortality is only 40 per cent without operation.

If the gut is empty its perforation closes by a local plastic process without general peritonitis.

Liver, spleen, and kidneys recover without much damage.

Hæmorrhage from injury to vessels is most common cause of death.

TREATMENT OF GUNSHOT WOUNDS.

EXPECTANT TREATMENT is the general rule.

AMPUTATE according to same rules as apply to lacerated wounds.

ARREST HÆMORRHAGE and immobilize fractures.

IN CRANIUM: Always trephine to remove splinters of inner table.

IN ABDOMEN: Do not explore unless signs of hæmorrhage or peritonitis occur.

Methods of Healing of Wounds.

HÆMORRHAGE fills interstices with clot.

THROMBOSIS closes ruptured vessels.

EXUDATION of leucocytes and serum from thrombosed vessels into edges of wound and blood-clot.

ABSORPTION of blood-clot and dead tissues by the leucocytes.

SUPPURATION.—If the wound is infected with a few micro-organisms of a mild type, these too are absorbed by the leucocytes. If, however, the infection is of a more virulent type, the leucocytes are killed, and form, with the serous exudation, pus.

FIBROBLASTS arise from endothelial cells and from fixed connective tissue cells, and line the edges or surface of the wound. They are large, elongated cells, with distinct nuclei.

Methods of Healing of Wounds, *continued.*

VASCULARIZATION of the layer of fibroblasts is brought about by a budding of solid outgrowths from the capillary walls. These outgrowths eventually become canalized, and joining other channels, form new capillary loops.

GRANULATION TISSUE is thus formed by a layer of fibroblasts vascularized by new capillary loops.

SURFACE OF WOUND heals by the growth of epithelial cells from the cut epithelial edges, over the summit of the granulation tissue.

CICATRIZATION of the wound is caused by conversion of granulation tissue into white fibrous tissue, the fibrillæ of which arise from the protoplasm of the fibroblasts, or as an intercellular exudation. Both nucleated cells and blood-vessels ultimately tend to disappear from the scar tissue owing to the contraction and compression of the fibrillæ.

SCARS OF RECENT WOUNDS will therefore be more vascular, i.e., redder, than surrounding parts in their early stages, when they consist of vascular granulation tissue. They will be less vascular, i.e., paler, than surrounding parts in their later stages, when they consist of avascular fibrous tissue.

CUTICLE COVERING SCARS is devoid of any skin structure or appendages. No hair, glands, papillæ, or lymphatics.

Modifications of Healing.

HEALING BY FIRST INTENTION.

- No gap is left between the divided wound surfaces.
- No loss of tissue by sloughing or septic infection.
- Granulation tissue is present as a thin layer only, between adjacent surfaces.
- Scar consists of a mere line of fibrous tissue.

HEALING BY SECOND INTENTION, or healing by granulation.

- A GAP IS LEFT between the divided edges of the wound, or
- A GAP IS CAUSED by the loss of tissue due to sloughing or septic infection.
    - When the edges of the wound have been sewn up, the depth of the wound is occupied by the dead tissues, either sloughs or pus, and further healing cannot occur until these have been removed by the opening of the wound.
    - Small aseptic sloughs may be absorbed or digested by the subjacent granulation tissue.
    - Large aseptic sloughs will be simply cast off.
    - Septic sloughs will be separated by a process of acute inflammation involving the loss of substance of more or less of the living tissues in the edges of the wound.
- THE GAPING FURROW between the wound surfaces has to be filled up by granulation tissue.

THE LARGE AREA OF GRANULATION TISSUE has to be covered over by an extensive sheet of cuticle.

The cuticle does not grow over the granulations unless the latter is on the same level as the edges of cut epithelial surface.

This "levelling up" is produced by the gap being filled with granulation tissue, and also by the contraction of the latter drawing the edges and the base of the wound towards one another.

THE SCAR resulting from this is a large mass of fibrous tissue in which much contraction takes place.

HEALING BY UNION OF GRANULATING SURFACES.

Produced when a gaping wound is brought together after it has become covered with granulations.

Granulating surfaces will unite if sepsis is absent, and if granulating surfaces are healthy and vascular.

HEALING UNDER A SCAB.—Healing by granulation when the granulating surface is covered by a mass of dried blood and exudation.

HEALING BY ORGANIZATION OF BLOOD-CLOT.

Large spaces are left in the depth of the wound which become full of blood-clot.

The blood is removed by leucocytes and these replaced by granulation tissue.

Blood only acts as a passive scaffolding for the activity of leucocytes.

Modification of Scars.

EXCESSIVE CONTRACTION produces great deformity when large subcutaneous areas are involved in the neck or flexures of limbs.

TREAT by dividing scar, stretching contracted part, and grafting new skin.

KELOID.—A vascular fibroid mass raised above the surface, and sending radiating processes in surrounding parts.

Specially frequent in tuberculous patients.

More often occurs when antiseptics are used than when these are avoided.

May be due to the involvement of a subcutaneous muscle (e.g., platysma) in the scar, constantly dragging upon it during healing.

Recurs on removal, but often disappears spontaneously.

ULCERATION.—Due to defective blood supply.

PAINFUL SCARS.—Due to implication of nerve terminals or a bulbous nerve end by contracting scar.

TREAT by opening and freeing or excising nerve.

MALIGNANT DISEASE may attack any scar. Generally affect superficial scars subject to much irritation, e.g., X-ray burns.

Slow-growing, fungating epithelioma.

Pain and lymphatic infection do not occur until neighbouring skin is invaded.

CHAPTER XI.

INJURIES OF BLOOD-VESSELS.*

HÆMORRHAGE.

Symptoms.

1. AFTER SUDDEN LARGE HÆMORRHAGE.
   - PALLOR of surface and of mucous membranes.
   - LIVIDITY exists if sudden death has followed wound of a large artery, because there has been no time for veins and capillaries to empty.
   - RESPIRATIONS quick, sighing and gasping.
   - PULSE rapid, small, and weak.
   - NOISES heard in ears.
   - DIMNESS OF VISION with flashes of light.
   - SYNCOPE, nausea, loss of consciousness.
   - RESTLESSNESS.
   - SURFACE IS COLD.
   - SWEATING is abundant.
   - SUBNORMAL TEMPERATURE.
   - GREAT THIRST.
2. AFTER RECURRENT SMALL HÆMORRHAGES.
   - ŒDEMA of eyelids and extremities.
   - LIABILITY TO FAINT on slight exertion.
   - PULSE is quick and easily compressible, with full beat and empty artery between beats, and well-marked dicrotic wave.

A TENDENCY FOR ALL THE SYMPTOMS TO INCREASE as bleeding continues.

Blood shows LEUCOCYTOSIS and diminution of red cells and hæmoglobin.

After hæmorrhage has ceased, especially concealed hæmorrhage, TEMPERATURE rises to 101°-102° F. for one or two days.

Circumstances which modify the effects of hæmorrhage.

QUANTITY of blood lost.

RATE of loss. The sudden loss of a comparatively small quantity is more dangerous than the gradual loss of a much larger quantity.

AGE of patient. Infants and aged patients bear loss of blood badly.

SEX. Women are less affected than men.

CONSTITUTIONAL CONDITION. The following render bleeding much more serious: Pre-existing ANÆMIA; any asthenic or cachectic disease; MARASMUS; co-existent SHOCK; SEPSIS.

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* Pearce Gould. Treves' "System of Surgery," Vol. I. i.

IN OPPOSITE CONDITIONS, notably in plethoric individuals with high tension pulse, or any cause of visceral congestion, cerebral or pulmonary in particular, BLEEDING MAY BE POSITIVELY BENEFICIAL.

Diagnosis of Hæmorrhage (especially concealed hæmorrhage).

- IN SHOCK.
  - SYMPTOMS are at their worst immediately after the injury, and TEND TO IMPROVE with time, except in fatal cases.
  - NERVE SYMPTOMS are much more prominent from the first: Unconsciousness, flaccid limbs, pupils dilated.
- IN CEREBRAL CONCUSSION.
  - Symptoms are of SUDDEN ONSET.
  - UNCONSCIOUSNESS is well marked and out of all proportion to the circulatory depression.
  - HISTORY of blow on the head.
- IN CEREBRAL COMPRESSION.
  - PARALYSIS and coma.
  - PULSE is full, strong, and slow.
  - RESPIRATIONS are deep and noisy.
  - SKIN is hot and congested.
  - PUPILS are often unequal.
- IN HÆMORRHAGE, on the other hand,
  - SYMPTOMS are trivial at first and increase in severity gradually.
    - Pallor, restlessness, dyspnœa, and thirst are conspicuous before unconsciousness occurs.
      - (For the diagnosis of abdominal, thoracic, and joint hæmorrhage, see the respective sections.)

The Arrest of Hæmorrhage always consists of two stages:—

- I.—TEMPORARY—The stopping of the bleeding. This can be NATURAL or ARTIFICIAL.
- II.—PERMANENT—The repair of wounded vessels. This is always a vital process, and nothing artificial can take its place.

The Natural Temporary Arrest of Hæmorrhage

- IS BROUGHT ABOUT BY
  - THE CONTRACTION of the ends of the vessel.
  - THE RETRACTION of the vessel in its sheath.
  - CURLING UP of inner coats within the outer.
  - COAGULATION of blood in and round the end of the vessel.
    - An internal clot forms between the nearest branch and the cut end of the vessel.
    - A central clot forms in the lips of the cut vessel.
    - An external clot forms outside the cut end of the vesse and between the middle coat and the sheath.
- IS AIDED BY
  - CONTUSION OR LACERATION of the vessel. This increases contraction and retraction of the coats.
  - DIMINISHED BLOOD-PRESSURE. Bleeding generally stops when the patient faints.

The Natural Temporary Arrest of Hæmorrhage—*continued.*

INCREASED COAGULABILITY of the blood. This occurs in anæmia; in asphyxia; after taking much milk, lime salts, or magnesium carbonate; when fluids are restricted.

IS RETARDED BY

CLEAN CUT wounds.

PARTIAL DIVISION of vessels, preventing contraction and retraction of vessel.

INCREASED BLOOD-PRESSURE: Renal disease; plethora; inflammation and congestion; exercise.

MOVEMENT of the part.

STIMULATION OF THE HEART (by drugs, alcohol, etc.).

HÆMOPHILIA.

CHRONIC JAUNDICE.

DIMINISHED COAGULABILITY OF THE BLOOD: Vegetarian diet; diminution of lime salts; citric acid; large quantities of fluid; restriction of food.

The Permanent Arrest of Hæmorrhage is produced by

HEALING OF THE WOUNDED VESSELS.

The clot becomes contracted and fissured.

The vasa vasorum dilate and exude plasma and white corpuscles.

The wall of the blood-vessel is thus swollen and more vascular.

The clot is penetrated by leucocytes and fibroblasts, the latter formed by proliferation of endothelium.

The red cells disintegrate and are removed by the leucocytes.

The fissures in the clot are lined by proliferating endothelium, thus forming new capillaries.

The capillaries communicate with (*a*) The lumen of the vessel; (*b*) The vasa vasorum.

The clot and the vessel wall eventually become organized into fibrous tissue.

DIFFERENCES IN HEALING OF ARTERIES AND VEINS.

IN AN ARTERY: A lateral wound does not heal until the lumen of the vessel is obliterated, because the high blood-pressure displaces the clot. When the lumen is once obliterated it seldom is opened up, because the thick arterial wall actively contracts and forms a strong scar.

IN A VEIN: A lateral wound, when once it is plugged by clot, heals. The blood-pressure is too low to disturb the clot. When a vein is obliterated by thrombosis, it often becomes re-canalized.

The Surgical Arrest of Hæmorrhage is brought about by closing the mouths of wounded vessels and holding them closed until they are plugged by firm clot. The following methods are employed:—

1. PRESSURE:—

AS THE PRELIMINARY to other means: On the artery at a distance from the wound. On the bleeding point.

As the Sole Means of arrest: Capillary or parenchymatous bleeding; bleeding from a superficial vein; bleeding in a bony cavity; bleeding from a hollow organ or cyst; deep hæmorrhage where ligature cannot be applied.

Its Dangers in other cases are: Inefficiency; gangrene conversion of open into concealed hæmorrhage.

2. FORCEPS: As a preliminary to ligature: As a sole means in small vessels. Cause adhesion of lips of vessel and curling up of inner coats.
3. CLAMPS.

   Rapid Method: A vascular pedicle is crushed and its vessels obliterated at one stroke, e.g., pedicles of polypi, appendix vermiformis.

   Slow Method: Clamps are left on for 48 hours. On inaccessible structures, e.g., pedicles of renal tumours or broad ligament of uterus.
4. TORSION. Vessel is twisted 8 half turns of the forceps. Inner coats curl up inside lumen. Outer coat twists up as a knot outside.
5. LIGATURE.—The method of choice in most cases (see "Ligature of Arteries").
6. CAUTERY.—Causes great contraction and retraction of vessels. A dull red heat only is essential, otherwise the vessel will be clean cut.

   Is Especially Useful: In parenchymatous bleeding; in bleeding from a sloughy surface; in internal piles.
7. COLD causes contraction of small vessels. Sucking ice for bleeding from mouth, throat, and stomach. Iced douches for epistaxis. Ice compress over a bleeding viscus; e.g., in hæmatemesis.
8. HEAT acts in the same way as cold. Hot water should be between 120° and 150° F. Useful for large bleeding surfaces, e.g., uterus, bone cavities, or cysts.
9. STYPTICS, e.g., alum, perchloride of iron, or tannic acid. They hasten coagulation of the blood. Very unreliable. Small wounds of the face, capillary oozing in mouth or nose, fungating tumours.
10. SUPRARENAL EXTRACT as a local injection or application. Injection up to 10 min. of 1–1000 adrenalin chloride. Causes contraction of small vessels. Is generally used in conjunction with eucaine as a preliminary to superficial operations.
11. ERGOT, administered by mouth or as a hypodermic injection. Used for post-partum hæmorrhage. Causes the uterus to contract as well as its vessels.

Treatment of the Effects of Hæmorrhage.

REST—local and general.

KEEP THE HEAD LOW—to prevent syncope.

WARMTH—By blankets and hot bottles.

Treatment of the Effects of Hæmorrhage—*continued.*

- INFUSION OF SALINE FLUID (sodium chloride ʒj to Oj).
  - INTO A VEIN.—Use 2–4 pints. Inject into a superficial vein, e.g., median basilic. Temperature 100° F. Avoid injecting air.
  - SUBCUTANEOUS.—Into the tissues under the mamma or axilla. Specially suitable for cases where assistance is unavailable.
  - CONTINUOUS TRANSFUSION.—The saline solution is allowed to continue running into the subcutaneous tissue, or into the rectum, slowly for 12–24 hours, using 6–10 pints of fluid.
- TRANSFUSION OF VASOCONSTRICTOR SOLUTIONS:
  - Adrenalin—Ergotinine.
    - Raise blood-pressure, and therefore combat shock. Are unsuitable if the hæmorrhage is likely to recur.
- RECTAL INJECTIONS.—One pint of hot water, with or without brandy, given every two hours (also continuous irrigation, see above).
- HYPODERMIC STIMULANTS.—Strychnine sulphate up to ♏ x of the liquor; ether ♏ xx–xxx. These are indicated for syncope; they have the disadvantage of tending to renew the bleeding.
- DIRECT STIMULATION of the heart by sinapism or fomentations.
- DIET.—Hot fluid nourishment at first; light and generous diet later.
- DRUGS.—Iron with quinine or nux vomica, for anæmia.
- GENERAL.—Prolonged rest, with plenty of sun and fresh air.

The Classification of Hæmorrhage.

- I.—ARTERIAL: (1) PRIMARY; (2) INTERMEDIATE or reactionary; (3) SECONDARY.
- II.—VENOUS.
- III.—CAPILLARY.

I. ARTERIAL HÆMORRHAGE

- OCCURS IN SPURTS synchronous with the heart's action.
  - Is bright red colour.
  - Is much more profuse from the proximal than from the distal end of the vessel.

1.—**Primary Hæmorrhage**—Occurs at the time of the injury.

- DUE TO a failure to occlude the wounded vessels.
    - It tends to become less and cease in time.
- TREATMENT.
  - ENLARGE THE WOUND AND SECURE THE BLEEDING VESSEL.—Except
    - Wounds in the tonsillar region, when ligature of the external carotid is indicated.
    - Stab wounds in palm and sole, where a graduated compress can be tried first.
  - TIE BOTH CUT ENDS in large vessels.
  - LET IT ALONE if the bleeding stops naturally, except :—
    - The distal end of a large artery.
    - A punctured wound of an artery.

2.—Intermediate or Reactionary Hæmorrhage

OCCURS WITHIN 24 hours of the injury.

DUE TO a failure of the means for the temporary arrest of hæmorrhage.

BROUGHT ABOUT BY :—

Patient's movements and restlessness, or rise of blood-pressure and increased force of the heart, which accompanies reaction after an operation.
The dislodgement of a clot from a vessel.
The slipping of a ligature.
Failure to secure the distal end of a wounded artery, which only bleeds after anastomising vessels have dilated.
Failure to occlude the lumen of a punctured artery. The clot is displaced from the punctured wound when blood-pressure rises.

TREATMENT.—The same as for primary hæmorrhage.

3.—Secondary Hæmorrhage

OCCURS LATER than 24 hours after the injury, especially between 7 and 10 days.

IS DUE TO failure of repair in a wounded vessel, or ulceration of a vessel.

CAUSES.

(*a*) SEPTIC INFLAMMATION of the artery.
(*b*) YIELDING OF AN ARTERIAL CICATRIX, brought about by division of its inner and middle coats by too tight ligature : disease of the artery, e.g., atheroma or calcareous change ; cachexia, hæmophilia, renal disease, diabetes ; violent cardiac action ; increased blood-pressure ; punctured wound of artery, the lumen of the artery not being obliterated.
(*c*) PROXIMITY OF LIGATURE TO A LARGE BRANCH, giving a very short cicatrix.
(*d*) WOUND OF A BRANCH just below the ligature, whose cicatrix gives way when full force of anastomotic circulation is established.
(*e*) FAULTY LIGATURE, which may be : Septic—Too tight—Hard knot or sharp edge—Too quickly absorbed, e.g., catgut.

PHENOMENA OF SECONDARY HÆMORRHAGE.—Is slight at first—Recurs at intervals—Becomes steadily greater at each recurrence—Has no tendency to spontaneous cure.

It is more frequent from the distal than proximal end of artery, because : at the distal side of a ligature there is smaller clot formed, the vasa vasorum are obliterated by ligature, the blood-pressure is higher.

TREATMENT OF SECONDARY HÆMORRHAGE.—It should always be dealt with, whether it has ceased or not, and whether it is small or large.

Secondary Hæmorrhage, *continued.*

(*a*) Pressure on Main Vessel above should be tried first.
(*b*) Open the Wound and ligature vessel above and below bleeding point. Endeavour to render the parts aseptic and drain.
(*c*) Cautery used at a dull red heat, in soft, sloughy wounds.
(*d*) Ligature of the Main Trunk above is indicated :—
  When above methods have failed.
  When it is known for certain what artery is bleeding.
  After amputation at shoulder or hip joints.
(*e*) Amputation is called for when the bleeding occurs from the femoral artery already ligated in continuity, because a second ligature would cause gangrene. In the arm a second ligature may be attempted.
(*f*) Firm Plugging is the only possible treatment in deep vessels of the root of the neck, groin, and abdomen.

II. VENOUS HÆMORRHAGE

OCCURS IN A STEADY STREAM, except
  From the cerebral sinuses it spurts synchronously with pulse.
  From large veins of the neck it spurts synchronously with respiration.
Is of a Dark Colour.
Is from the Distal end of the divided vessel only ; except : in the neck, in varicose veins, in branches of large veins, if no valve intervenes.

TREATMENT.
  Pressure is sufficient in superficial veins.
  Ligature is needed for veins in the neck—Deep veins—Varicose veins.
  Plugging is necessary when veins lie in bony cavities, e.g., cranial sinuses.
  Suture, or Ligature, in punctured wounds of large veins, so as not to occlude the lumen.
  Tie the Artery at a different place, but do not occlude the vein, if main vein of a limb is wounded slightly when operating to ligate the main artery in continuity.

III. CAPILLARY HÆMORRHAGE

(including parenchymatous hæmorrhage) consists of an oozing from wound surfaces.

TREATMENT.—Sew up the wound ; or, when this is impracticable, pressure, plugging, or the cautery must be used.

HÆMOPHILIA.

DEFINITION.—A hereditary disease in which there is a tendency to spontaneous hæmorrhages and very profuse and uncontrollable bleeding from wounds.

INHERITANCE.—The disease is peculiar to the male sex, but is transmitted by females ; thus the sons develop hæmophilia, and the daughters who are free from the disease often beget hæmophilic sons.

PATHOLOGY.—Of this little is known. Probably the seat of morbidity is in the walls of the small vessels, which are so constituted that intravascular clotting does not take place. The blood clots in the usual time after it has been shed.

TRAUMATIC HÆMORRHAGES.—Except that the division of the umbilical cord is remarkably free from incident, every trivial wound is accompanied by steady and persistent oozing. Tooth extraction is often the first injury to call attention to the disease, and the steady bleeding from this may be fatal. Circumcision, or any other operation, will cause dangerous or fatal bleeding. The actual hæmorrhage is continued steadily as a capillary oozing for many days, and it is the consequent anæmia that causes death by syncope.

ECCHYMOSIS.—Slight or unnoticed trauma produces extensive subcutaneous ecchymosis; hence the sufferer is often called "a bruiser and bleeder." The bruises slowly disappear if no external wound exists.

JOINTS.—Bleeding into the joints is common, and produces changes of synovitis and osteo-arthritis eventually (see Chap. XXII).

MUCOUS HÆMORRHAGES.—The mucous membranes are not so prone to bleeding as might be anticipated. But epistaxis, hæmatemesis, or melæna occasionally occur in a severe or fatal form.

COURSE.—In bad cases the subjects seldom survive to maturity, but in many instances the tendency to bleeding becomes less with advancing years.

TREATMENT.—From a surgical point of view it is emphatically negative. It is most important to avoid performing operations on hæmophilic subjects, and when hæmophilic symptoms are present to abstain from any active measures such as the cautery or aspiration of blood effusions.

Prolonged rest and firm pressure are the only methods ordinarily available.

Calcium chloride should be given in large doses by the mouth.

OPEN WOUNDS OF BLOOD-VESSELS.

These are dealt with under the subject of Hæmorrhage, and it only remains to note—

Incised Wounds bleed very freely, because there is but little injury to cause the inner coats to curl up.

Incised wounds which do not completely sever the vessel bleed most of all, because both retraction and contraction are prevented.

Lacerated Wounds bleed less than any, or not at all, because the inner coats are curled up by the violence of the injury, and the outer sheath is also twisted up as in torsion.

Punctured Wounds—as when a bullet nicks one side of an artery—may result in a traumatic aneurysm; or, if the adjacent vein is wounded at the same time, an arterio-venous aneurysm, or aneurysmal varix, will result.

TREATMENT of wounded arteries and veins.

LIGATURE both ends of divided vessel, or above and below a lateral wound.

EXCEPTIONS.—Large veins should have lateral wounds carefully sutured. Large arteries may also have lateral wounds sewn together with fine silk—but this is not the usual practice. The proximal end of a divided artery may be sewn to the distal end, so preserving the lumen.*

SUBCUTANEOUS INJURIES OF BLOOD-VESSELS.

Contusion and Laceration of Arteries.

CAUSES.—External blow, being run over, etc. Counter-pressure when reducing an old dislocation. Bullets of low velocity.

PREDISPOSING CONDITIONS.—Atheroma, or calcareous degeneration.

RESULTS.

SLIGHT INJURY TO THE INNER COATS, with proliferation of the intima and thrombosis.

RUPTURE OF INNER COATS, with thrombosis, aneurysm from yielding of the outer coat, dissecting aneurysm.

INJURY WITH SUBSEQUENT SLOUGHING OF THE OUTER COAT, producing secondary hæmorrhage or aneurysm.

INJURY TO THE VESSEL MAY BECOME INFECTED, causing septic arteritis, secondary hæmorrhage, or aneurysm.

RUPTURE OF ALL THE COATS of the artery, with immediate thrombosis, or, more commonly, diffuse traumatic aneurysm.

GANGRENE may follow a contusion of an artery: from simple thrombosis, especially if the artery is calcareous; rupture of the artery; diffuse aneurysm.

SYMPTOMS OF THROMBOSIS.—Loss of pulsation in the vessels beyond the injury. Possibly a tender swelling in the position of the injured vessel.

These may appear at once after the injury, indicating rupture of the inner coats; or may be delayed for several days, indicating thrombosis from contusion and proliferation of the intima.

SYMPTOMS OF ANEURYSM.—A pulsating swelling connected with the artery appears from a few days to a few months from the time of injury. Common mode of origin of axillary and popliteal aneurysms.

SYMPTOMS OF RUPTURE, with the formation of a "diffuse aneurysm":—

LOCAL SIGNS: Sudden, sharp pain at the seat of injury. Patient may be conscious of a snap. Fusiform swelling rapidly appears at injured point. Pulsation and bruit sometimes present, often not.

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* Various papers and references in "Annals of Surgery," 1907, xlvi.

DISTAL SIGNS: Cessation of pulsation in vessels beyond. Limb is cold and numb. Lividity and œdema from venous obstruction.

GENERAL SIGNS: Weak, rapid pulse. Pallor, dyspnœa, and syncope.

TERMINATION OF RUPTURE.

(*a*) RECOVERY.—The swelling becomes hard by coagulation. The ruptured ends of the vessel heal in the ordinary way. Collateral circulation is established.

(*b*) GANGRENE is produced by the pressure of the blood obliterating both the veins and collateral circulation when the main trunk is already severed.

(*c*) EXTERNAL RUPTURE through the skin, and death from external hæmorrhage.

(*d*) SUPPURATION of the blood mass, with subsequent bursting of the abscess and death from secondary hæmorrhage.

(*e*) DEATH from internal hæmorrhage. Especially if the ruptured vessel communicates with some internal cavity.

TREATMENT OF SUBCUTANEOUS INJURIES OF ARTERIES.

1. IF THE SIGNS POINT TO THROMBOSIS:—

   Rest—Warmth—Aseptic preparation of the skin.

2. IF THE SIGNS POINT TO RUPTURED ARTERY:—

   Put on a tourniquet above the injury if possible.

   Cut down on the swelling and turn out the clots.

   Tie both ends of the ruptured vessel. (It is possible to sew the proximal end of the vessel to the distal without obliterating the lumen. This is not, however, often attempted.)

3. IF THE SIGNS POINT TO SUPPURATION:—

   If a tourniquet can be applied, proceed as in 2.

   If a tourniquet cannot be applied, tie the artery above the injury before opening the abscess.

4. AMPUTATION is necessary if gangrene or secondary hæmorrhage occurs.

CHAPTER XII.

THE DISEASES OF ARTERIES.

INFLAMMATION AND DEGENERATION OF ARTERIES.

Varieties of Arteritis.

ACUTE ARTERITIS.

SIMPLE OR PLASTIC.—Producing the repair of wounded arteries.

SEPTIC.—Resulting from septic injuries and wounds.

EMBOLIC.—Resulting from the lodging of an infective embolus in the lumen of an artery.

CHRONIC ARTERITIS.

ENDARTERITIS DEFORMANS.—Atheroma. Chronic inflammation of the inner coats, of patchy distribution, affecting large vessels and resulting in dilatation.

ENDARTERITIS OBLITERANS.—IDIOPATHIC, SYPHILITIC, TUBERCULAR, DIABETIC.—Uniform inflammatory proliferation affecting the whole circumference of small vessels and resulting in occlusion.

Simple, Plastic or Traumatic Arteritis.

PRODUCED by aseptic contusions and wounds.

RESULTS in proliferation of the tunica intima—Exudation from the vasa vasorum—Thrombosis of the vessel and conversion into a fibrous cord.

Is to be regarded as a conservative act of repair rather than disease.

Septic Arteritis.

CAUSES : A septic wound—A septic ligature—The extension of an abscess—Very virulent ulceration, e.g., phagedæna. (Infection from the blood-stream is considered under the next head.)

PATHOLOGY.—The coats of the artery become acutely inflamed.

They are softened and disintegrated by the peptonizing action of bacteria and their toxins.

The leucocytes and fibroblasts of the exudation are killed by the toxins, and form pus.

The blood-clot in the vessel is either disintegrated by the same agents, or washed out by the blood-stream.

RESULTS.—Secondary hæmorrhage (*vide supra*), Hæmorrhage from phthisical cavities, from chronic ulcers, or from malignant growths, is generally the result of this process.

TREATMENT.—That for secondary hæmorrhage, etc.

Embolic Arteritis.—Generally in young people. An infective embolus is dislodged from the aortic valves in malignant endocarditis or acute rheumatism. It blocks a small artery.

RESULTS: (1) An abscess, as in pyæmia (2) An idiopathic aneurysm—An acute softening of the arterial wall yields to the blood-pressure, and so gives rise to an aneurysm.

Atheroma.

CONSISTS IN a chronic endarteritis, which results from long-continued strain, and produces degeneration of the arterial wall.

CAUSES: Old age—Mechanical strain of laborious occupations—Syphilis—Chronically raised blood-pressure: e.g., renal disease, gout, lead poisoning, alcoholism.

MORBID ANATOMY, ETC.

Distribution: Aorta—Large arteries—Coronary arteries—Arteries at the base of the brain—Splenic artery—Arteries of the leg more frequently than the arm—The curved arteries and the convex side of the curved portions are first affected—Points where large arteries bifurcate—Points where branches are given off from large vessels—Points where arteries curve round bones, e.g., the subclavian over the first rib—Points where arteries are subject to constant flexion, e.g., the popliteal.

NAKED-EYE APPEARANCE.

Vessel is Dilated and Hypertrophied. In some cases elongated and tortuous.

Interior Presents: Opaque oval white patches under the endothelium—Calcareous plaques bared of endothelium—Softened white swellings, the atheromatous abscesses—Shallow excavations into the tunica media, atheromatous ulcers.

MICROSCOPICAL CHANGES.

Proliferation of the cells of the deep layer of the tunica intima.

This tissue is avascular, and soon degenerates:—

Fatty degeneration, or calcareous degeneration forming "laminar calcification" of the artery.

The tunica media is invaded to some extent.

The endothelial lining cut off from the vasa vasorum is lost.

The tunica adventitia is thickened.

RESULTS OF ATHEROMA.

1. Aneurysm—from yielding of the weakened wall.
2. Dissecting Aneurysm—by a forcing of the blood between the layers of an artery at the edges of an "atheromatous ulcer."
3. Thrombosis—possibly gangrene.
4. Rupture from slight violence.
5. Embolic Obliteration of the vessel by a detachment of a calcareous plate.
6. Degeneration of Structures Supplied, e.g., in the case of the heart (fibroid or fatty) or the brain (white softening).

TREATMENT.—Light diet, with little meat and no alcohol. Gentle exercise. Keep the blood-pressure low by aperients or nitrites.

Syphilitic Endarteritis.—Occurs in the tertiary stage of the disease. Attacks small arteries. Brain, kidneys, and the region of gummata are most commonly affected.

CONSISTS IN: A proliferation of the cells of the tunica intima. The new layer of cells is vascularized from the vasa vasorum. Slight thickening of the tunica media. Some thickening of the adventitia.

RESULTS IN: Obliteration of the lumen of the vessel. Degeneration of the tissues supplied into gummatous material, or, in the brain, white softening with consequent paralysis.

TREATMENT.—Iodide of potassium.

Endarteritis Obliterans.—Produces similar changes to the last described. There is no evidence of syphilis. Coldness, numbness, and gangrene of the parts supplied. Great pain.

Tuberculous Endarteritis.—Always occurs in the area invaded by tubercle. The small vessels are obliterated by a proliferation of the intima. Hence tuberculous areas undergo caseous degeneration.

(The ulceration which opens large vessels and causes serious hæmorrhage is always produced by secondary sepsis, e.g., in the lung or intestine.)

Diabetic Endarteritis.—Similar to the above obliterating forms of endarteritis. Affects the anterior and posterior tibial arteries most commonly. Produces one form of diabetic gangrene.

Degeneration of Arteries.

FATTY DEGENERATION.—Affects the intima—Occurs in the aorta—Is difficult to distinguish from atheroma.

CALCAREOUS DEGENERATION.—Occurs in old people—Affects the tunica media—Muscle fibres are transformed into rings of calcified material—Occurs in medium-sized arteries, e.g., brachial.

PRODUCES: Loss of arterial tone—Deficient circulation in the parts supplied—Coldness and numbness—Cramps, pain and tingling—Thrombosis—Senile gangrene (the branches are calcified, and are therefore unable to dilate so as to form efficient collateral circulation)—Rupture from slight violence—Secondary hæmorrhage after operations.

* ANEURYSM.

Definition.—A blood-tumour communicating with an artery.

Classification.

I.—IDIOPATHIC, or spontaneous: (1) FUSIFORM; (2) SACCULATED; (3) DISSECTING.

II.—TRAUMATIC: (1) CIRCUMSCRIBED; (2) DIFFUSE, or ruptured artery.

III.—ARTERIO-VENOUS: (1) ANEURYSMAL VARIX; (2) VARICOSE ANEURYSM.

IV.—ANGEIOMATA: (1) CIRSOID ANEURYSM; (2) ANEURYSM BY ANASTOMOSIS.

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* Pearce Gould. Treves' "System of Surgery," Vol. I.

I.—IDIOPATHIC ANEURYSM.

Causes.

(*a*) WEAKENING OF THE ARTERIAL WALL: Atheroma—Syphilis (?)—Embolic arteritis—Partial laceration, or the cicatrix of an old injury.

(*b*) INCREASE OF THE BLOOD-PRESSURE: Violent exercise, especially when intermittent—Cardiac hypertrophy—Plethora—Chronic renal disease—Gout and lead poisoning.

(*c*) INDIRECT CAUSES, which bring about I or II, or both.

AGE.—That of maximum activity—30–50. Aneurysms due to infective emboli are the only ones which occur more commonly in children.

SEX.—Males are ten times more liable than females, except in carotid aneurysm and dissecting aneurysm, which are commoner in women.

OCCUPATION.—Soldiers, sailors, athletes, etc.

NATIONALITY.—Anglo-Saxons.

STRAIN.—Right arm is more prone than the left. Vessels which are liable to flexion and extension, e.g., the popliteal, are very liable to aneurysm.

ALCOHOLISM.

SYMMETRY, as seen, e.g., in double popliteal aneurysm, is caused by identical conditions on the two sides.

1. Fusiform Aneurysm.

PATHOLOGY.—The weakened arterial wall yields in every direction to the blood-pressure.

The vessel is thus elongated and dilated in its entire circumference.

All three coats of the vessel form the aneurysm.

The wall of the aneurysm is thicker than that of the normal artery.

The inner coat is generally thickened by atheroma.

The middle coat is thinned.

The outer coat is thickened by fibrous tissue.

Contains little or no clot.

DISTRIBUTION.—Aorta (common)—Largest arteries (rare).

COURSE.—Of slow development and progress. Comparatively symptomless. It may fill with clot, and be thus cured.

COMPLICATIONS.—One portion of the sac wall gives way, and a sacculated aneurysm is produced.

TREATMENT.—Only constitutional.

2. Sacculated Aneurysm.

PATHOLOGY.—One point in the arterial wall is weaker than the rest. This may be a thin patch of atheroma, or a scar, or an "atheromatous ulcer." A local bulging takes place at this spot. The inner and middle coats disappear in this situation.

THE OUTER COAT (added to by fibrous tissue) forms the only layer of the sac.

Sacculated Aneurysm, *continued.*

The Absence of the Inner and Middle Coats is of the utmost importance.

They form three-quarters to seven-eighths of the thickness of a normal artery.

They contain most of the elastic and muscular tissue to which the vessel owes its resiliency.

They contain the vasa vasorum, to which the vessel owes its nourishment.

The Sac is Strengthened

From the outside, by the addition of fibrous tissue and by the incorporation of surrounding structures.

From the inside, by the deposition of laminated clot.

CONTENTS. (1) White, laminated, fibrinous clots at the periphery. Generally in the parts least exposed to the blood-stream.

This is seldom organized into fibrous tissue, because of constant movement of pulsation, and absence of the tunica intima with vasa vasorum.

(2) Red blood-clot of recent origin may be present, always when the aneurysm has lately been cured.

(3) Fluid blood communicating with blood-stream.

RELATION OF THE CONTENTS TO THE SAC WALL.—The presence of laminated clot strengthens the sac. The presence of any clot lessens the fluid contents.

The pressure on the sac wall of an aneurysm depends on the area of fluid blood it contains, and varies as the square of the diameter of the fluid-containing cavity. It is thus a "living Bramah press."

Effects of an Aneurysm.

A.—ON SURROUNDING STRUCTURES.

Excites Inflammatory Reaction.—Usually plastic. When infected it may be suppurative.

Veins.—Compression, with cyanosis and œdema. Rarely a communicating perforation is formed.

Nerves are flattened out and destroyed. Great pain—rarely numbness and anæsthesia. Motor paralysis (rare, except in the case of the recurrent laryngeal).

Bones are absorbed and eroded : notably the vertebræ and sternum.

Cartilages are much more resistant than bones.

Tendons and Fasciæ are incorporated in the sac.

Mucous Canals are compressed and displaced.

B.—ON THE CIRCULATION.

Loss of Force, i.e., of blood-pressure beyond the aneurysm.

Partly compensated by cardiac hypertrophy and dilatation of anastomotic channels.

The Pulse.—Is delayed. Is smaller and weaker. The impulse and dicrotic waves are absent.

An Artery may be Blocked by the cure of the aneurysm ; by the pressure of the aneurysm on the vessel ; by the involvement of a branch of the main vessel in the sac.

Course and Terminations.—(1) CURE; (2) GROWTH AND RUPTURE; (3) SUPPURATION AND RUPTURE (or cure—very rare).

CURE OF AN ANEURYSM occurs in three stages:—

1. FILLING OF THE SAC with blood-clot.
2. OBLITERATION OF THE LUMEN of the vessel between the aneurysm and the nearest collaterals. This is the essential condition of cure without which the final stage cannot occur.
3. ORGANIZATION OF THE BLOOD-CLOT into fibrous tissue.

SPONTANEOUS CURE may be caused by:—

Extension of the blood-clot from the aneurysm into the artery.

Pressure of the aneurysm upon the artery so as to obliterate it.

Displacement of laminar clot from the aneurysm into the artery as an embolus.

Arteritis excited by mere irritation (plastic), or suppurative by infection. Both of these are very rare.

RUPTURE OF AN ANEURYSM is much the commonest natura termination.

THROUGH THE SKIN OR MUCOUS MEMBRANE.

By small ulcer at first.

Bleeding, like that of secondary hæmorrhage.

Externally, œsophagus, trachea, bronchus, stomach, intestine

THROUGH SEROUS OR SYNOVIAL MEMBRANE.

By slitlike rupture.

Immediate large, generally fatal hæmorrhage.

Pleura, pericardium, knee joint.

SUBCUTANEOUS RUPTURE.—Blood may extend without definite limit, producing gangrene as in a ruptured artery. Blood may be circumscribed for a time: the so-called "leaking aneurysm."

SUPPURATION OF AN ANEURYSM.—Caused by infection of the tissues in and round the aneurysm.

Their resistance may have been weakened by prolonged or rough pressure or manipulation, or sudden coagulation in a large sac.

It is especially common in axillary aneurysm.

The sac wall sloughs.

The artery above and below bleeds furiously when the abscess opens.

Rarely the artery above and below is sealed by plastic arteritis, and the aneurysm is thus cured.

Signs of a Sacculated Aneurysm.

TUMOUR.—Placed over and fixed to an artery. Pulsates synchronously with the heart.

Pulsation is expansile, and ceases when the artery above is compressed.

A BRUIT is heard over the swelling, and sometimes a thrill is felt.

Systolic in time, and blowing in character. Increased by pressure of the stethoscope. Rarely it is diastolic as well as systolic, especially in aortic aneurysms.

Signs of a Sacculated Aneurysm, *continued.*

PULSE BEYOND is delayed in time and lessened in force.

PRESSURE SIGNS: Congestion of veins with œdema. Paralysis of nerves. Muscular wasting.

PAIN.—Constant severe boring, especially when bones are eroded. Occasional lancinating pains in area of distribution of sensory nerves.

Diagnosis.

ABNORMAL PULSATING VESSELS, e.g., "Pulsating Aorta." In this there is no tumour.

TUMOUR OVER AN ARTERY.—In this the pulsation is heaving but not expansile; the pulsation ceases if the tumour is lifted off the artery; pressure on the artery does not affect the size of tumour.

A bruit is sometimes heard, but is rasping and only occurs along the line of the vessel.

CYSTS OVER ARTERIES, which may communicate with joints.

Pulsation may be present and may be expansile. But pressure on the artery above does not lessen the size of the tumour.

If the cyst can be emptied into the joint, it fills out gradually and not by jerks.

PULSATING VASCULAR TUMOURS, e.g., Goitre or Sarcoma.

Tumour cannot be much reduced either by pressure or by compressing the artery.

Bruit is whiffing, and very variable in different parts.

The tumour can often be moved away from or along the main artery (e.g., goitre).

The tumour is often in a situation where no large artery exists (e.g., the front of the head of the tibia).

Modifications of the Signs of an Aneurysm.

ANEURYSM UNDERGOING CURE.—The sac is partly filled with clot. The pulsation loses its expansile character. Pulsation ceases when the lumen of the artery is obliterated.

LEAKING OF RUPTURED ANEURYSM.—Rapid increase in size. Outline is indefinite. Pulsation and bruit are lessened or disappear. Pain and collapse. Congestion, œdema, and numbness of parts below.

SUPPURATING ANEURYSM.—Increase in swelling, with indefinite outline. Pulsation and bruit lessened. Signs of acute inflammation. Œdema over tumour.

Treatment of Aneurysm.

CONSTITUTIONAL.

Absolute rest, mental and physical.

Starvation, especially as regards liquids: Bread 6 oz., meat 2 oz., milk 6 oz. daily.

Large doses of potassium iodide to reduce blood-pressure.

LOCAL.

- (i) COMPRESSION of the artery above the aneurysm.
  - Digitally. By relays of assistants acting 20 min. at a time. The thumb is reinforced by a 6-lb. weight.
  - Instruments, e.g., the various tourniquets.
  - Results of compression: Often nothing, or
    - Aneurysm may be gradually cured by deposition of laminated clot.
    - Aneurysm may be suddenly cured by clotting of all the blood in the aneurysm and vessel at once.
    - The condition often recurs from a dissolution of the clot before organization has taken place.
  - Disadvantages of compression: Very painful, requiring anæsthetic or opiates. Digital requires so many assistants. Instrumental often causes sloughing of the skin. Is very tedious and uncertain. It causes enlargement of collaterals, and therefore subsequent cure by distant proximal ligature is rendered uncertain.
- (ii) FLEXION OF ELBOW OR KNEE.—Only applicable to aneurysms at the elbow or ham when these are of small size.
- (iii) ESMARCH'S BANDAGE.—Applied for $1\frac{1}{2}$ hours under an anæsthetic. Followed by slight compression of the artery above for some days.
  - Is very uncertain. It may rupture the sac or cause thrombosis of the collaterals, with consequent gangrene.
- (iv) LIGATURE OF THE MAIN ARTERY—the routine method.
  - (*a*) Proximal. Close to the sac (Anel's operation); at a distance from the sac (Hunter's operation).
  - (*b*) Distal. Used when proximal side of artery is out of reach. Main vessel: (Brasdor's); Main branches of the vessel (Wardrop's).
  - The above places of ligature are placed in the order of efficiency (*see below*).
- (v) EXCISION OF ANEURYSM. Preliminary tourniquet. All vessels connected with the sac are ligatured and cut. Sac is carefully dissected out.
- (vi) INCISION OF ANEURYSM * (Matas' modification of the "old operation"). Preliminary tourniquet. Sac is freely opened and emptied of blood and clot. The mouths of all vessels opening into the sac are sewn up by separate silk sutures. The redundant portions of the sac are removed. The rest of the sac is folded over and sewn together as a solid pad under the skin.
  - The use of stitches from the inner side is the distinctive feature of this operation.
  - Advantages over excision are, no risk of injury to veins, nerves, and collaterals which lie so close to the sac.
  - The only method available when the proximal ligature has failed.

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* "Medical News," 1888, liii; "Annals of Surgery," 1903, xxxvii; Ibid, 1907, xlvi.

Treatment of Aneurysm, *continued.*

(vii) The Introduction of Foreign Bodies into the Sac.—Only used in inoperable situations as in the aorta, and in the case of the abdominal aorta it has given recent successes: (*a*) Simple needles; (*b*) Needles connected with the positive pole of a battery; (*c*) Piano wire: many feet passed into the sac and left. May be passed at random through a cannula. Best passed by a special (Colt's) mechanism, as a complete spherical wire network.

(viii) The Subcutaneous Injection of Gelatin.—10–20 oz. of a sterilized solution of gelatin is injected into the subcutaneous tissue. Only used in internal aneurysm.

(ix) Amputation.—Ruptured or inflamed aneurysms (in certain cases). When gangrene or secondary hæmorrhage have resulted from ligature. Subclavian aneurysm (rare).

ON THE CHOICE OF THE METHOD OF TREATMENT.

Matas' Operation is the ideal treatment, and the only one when ligature has failed.

Proximal Ligature close to the Sac is simple and efficient, but sometimes difficult because of the altered anatomical relations.

Proximal Ligature at a Distance from the Sac is easy, but liable to failure if the collateral circulation is too free, or to gangrene if it is not free enough.

Compression is to be used: In very nervous people who dread an operation; in patients with severe heart disease; in patients with extensive disease of the vessels.

The other methods are only suitable for special cases.

On the Ligature of an Artery in Continuity.

LIGATURE MATERIALS.

Silk.—Can be sterilized by boiling, but is unabsorbable, and therefore, if it become septic, may cause a sinus. It is the most generally used ligature.

Catgut.—Requires complicated sterilization and it is too quickly absorbed to be trusted for large arteries.

Kangaroo Tendon and Ox Aorta.—Are absorbed more slowly than catgut.

METHOD OF TYING.

Stay Knot should be used by taking two strands of ligature, tying the first turn of a knot in each separately, and then knotting the two pairs of ends together.

Tightly enough to occlude the lumen without rupturing the inner coats of the vessel.

Ligature should be passed inside the sheath of the vessel. The vessel should be disturbed from its bed as little as possible.

CHANGES PRODUCED IN THE ARTERY.—The lumen is occluded. The inner and middle coats are ruptured and curl up inside the vessel if the ligature is drawn too tight. Clot forms between the ligature and nearest branches above and below. Clot on the distal side of ligature is smaller and slower in formation than on the proximal. A portion of the occluded vessel is converted by the organization of the blood-clot into a fibrous cord.

ADVANTAGES OF APPLYING THE LIGATURE WITH MODERATE FORCE.—Inner and middle coats are not ruptured. Less liability to secondary hæmorrhage, especially when the vessel is diseased or when the ligature is very near a large branch. Vessel walls are much thinner when distended by blood than after death. Outer coat is very thin, only $\frac{1}{4}$ to $\frac{1}{25}$ of the whole thickness.

LIGATURE FAILS TO OCCLUDE when: (1) Knot has slipped—In single-strand ligatures the beat of the artery opens the first turn of the knot whilst second is being tied. (2) Ligature is absorbed before organization of the clot has occurred.

EFFECTS ON THE CIRCULATION.—The heart is embarrassed when a large artery is ligatured. Parts below the ligature become cold and pulseless; then the anastomotic channels dilate. Pulsation below the ligature is gradually resumed. Tissues become hyperæmic before they are natural. Some of the anastomotic channels permanently hypertrophy.

TREATMENT AFTER LIGATURE.—Parts should be kept warm and at rest in a horizontal position. Any tight bandaging should be avoided.

GANGRENE MAY OCCUR AFTER LIGATURE.

CAUSES. (1) Circulation being too weak to open up anastomotic channels, either from a weak heart or as the result of a large hæmorrhage. (2) Diseased vessels unable to dilate for anastomosis. (3) Vessels contained in bony canals, e.g., interna carotid and vertebral, cannot dilate. (4) Pressure of a tight bandage on anastomosing circulation. (5) Thrombosis extending down the vessels. (6) Injury to the main vein. (7) Cold or inflammation occurring directly after ligature.

EXTENDS.—Generally as dry gangrene of only one or two toes, or dry gangrene up to the ligature; or sometimes as moist gangrene up to the ligature.

TIME.—Occurs third to tenth day after ligature.

TREATMENT.—Limited dry gangrene: wait to see the extent of natural repair. Extensive gangrene—dry or moist: amputate.

PROXIMAL LIGATURE CLOSE TO THE SAC (Anel's operation) causes obliteration of the artery and extension of the clot from the ligature to the sac.

It is most efficient because no branch intervenes between the ligature and the sac. It only causes one block in the circulation, and therefore only one set of anastomosing vessels are required. It is difficult to perform, because the aneurysm so distorts the anatomical relations.

PROXIMAL LIGATURE AT A DISTANCE FROM THE SAC (Hunter's operation) is easy to perform. The artery is more likely to be healthy.

Causes a double block in the circulation; (1) At the ligature extending to the next branch; (2) Blood clots in the sac and extends to the vessel from which it springs.

On the Ligature of an Artery in Continuity, *continued.*

Circulation has to be carried from the artery above the ligature by anastomotic channels to the artery between the ligature and the aneurysm. Thence by a second set of anastomosing vessels to the artery below the aneurysm. Hence two sets of anastomoses are required, and gangrene may arise by a failure of this double set.

Failure to cure occurs in Hunter's operation from too great freedom of anastomosing channels (this is often the case after long compression); the blood does not clot in the sac.

SIGNS AFTER HUNTER'S OPERATION when successful.—Shrinking and loss of pulsation in sac. Return of slight pulsation when the first anastomosing channels have dilated. Gradual consolidation of the sac as the second anastomosis is established.

Dissecting Aneurysm.

ETIOLOGY.—A rare disease, commoner in women than men.

ANATOMY.—Begins in the aorta and spreads down to the iliac arteries and up the carotids. The blood is forced between the coats of the vessels, separating the inner and middle from the outer. It probably begins at an atheromatous ulcer. The folded inner coats block the lumen of the vessels.

SYMPTOMS.—Sudden, severe pain in the back and trunk, with coldness and pain in the legs from cessation of the circulation.

TREATMENT is impossible, and a fatal result occurs in a few days.

II.—TRAUMATIC ANEURYSM.

CAUSES.—(1) Contusion or subcutaneous rupture; (2) A small punctured or valvular wound of an artery; (3) The yielding of a cicatrix of a partially divided artery; (4) Closure of the external wound over a wounded artery.

DEGREES.—(1) Circumscribed; (2) Diffuse.

Circumscribed Traumatic Aneurysm.—The aneurysm is limited by a distinct sac. It is usually formed by the yielding of a cicatrix, or by the relaxation of pressure over a vessel some time after the injury. Its course is that of a spontaneous aneurysm.

OCCURS most commonly in the hand, foot, or scalp.

TREATMENT.—Pressure, excision, or ligature.

Diffuse Traumatic Aneurysm.—Occurs soon after the injury, and the blood collection is widely diffused in the fascial planes without a limiting sac.

SIGNS.—A rapidly growing swelling in the line of an artery. Pulsation and bruit are ill-marked. The skin is discoloured and tightly stretched. The pulse is weak or lost below. Pain is intense.

RESULTS.—(1) External bursting; (2) Gangrene; (3) Suppuration.

DIAGNOSIS has to be made from abscess or cellulitis by the thrill and bruit and great interference with the circulation. Also by its rapid development.

OCCURS most commonly in the axilla, groin, and ham.

TREATMENT.—Incision and ligature above and below the wound.

III.—ARTERIO-VENOUS ANEURYSM.

Definition.—An abnormal communication between any artery and vein.

VARIETIES.—(1) Aneurysmal varix; (2) Varicose aneurysm.

Aneurysmal Varix.—A dilated varicose vein communicating directly with an artery.

CAUSES.—Stabs, phlebotomy, or gunshot wounds, which simultaneously wound a vein and artery. Very rarely idiopathic or congenital. Most common at the elbow.

STRUCTURE.—The artery is enlarged and thickened, the vein is tortuous, and its branches varicose, with a large sac opposite the communication with the artery.

SYMPTOMS AND SIGNS.—A fluid compressible swelling associated with varicose veins. Marked pulsation and a loud bruit are evident in the swelling, which partly empties on raising the limb. Pain may be severe, and congestive signs may occur in skin below.

TREATMENT.—Pressure or ligature of the artery, or suture of the opening in the arterial wall.

Varicose Aneurysm.—An aneurysm which communicates with a vein as well as with an artery.

CAUSES.—As above, and also by the pressure of an ordinary aneurysm opening into a vein.

SIGNS are the same as in the last, except that a more solid aneurysmal tumour may be detected in addition to the pulsating varicose veins.

TREATMENT.—Excision, with ligature of artery and vein.

(Class IV is more conveniently considered with other angeiomata.)

CHAPTER XIII.

DISEASES OF THE VEINS & LYMPHATICS

VENOUS THROMBOSIS.

Definition.—A coagulation of the blood in the veins.

Causes.

INJURIES OF THE VEIN WALLS: Contusion, laceration, compression, ligature.

INFLAMMATION OF THE VEIN: Phlebitis. All septic processes are apt to cause phlebitis and then thrombosis in neighbouring veins, e.g., appendicitis often causes femoral thrombosis.

DEGENERATION OF THE VEIN: Varicose veins. The dilated cavities, rough coat, and sluggish stream often produce thrombosis.

BLOOD CHANGES. DIMINISHED RATE OF FLOW. (*See* "Factors Promoting Coagulation.")

Varieties of Thrombi.

RED THROMBI are formed by clotting *en masse*, so that the red cells are included.

WHITE THROMBI consists of fibrin slowly formed from which the red cells are absent.

INFECTIVE THROMBI contain living micro-organisms.

Effects of Thrombosis on the Vein.

(1) The clot may become organized into connective tissue and the channel permanently occluded.
(2) The clot after becoming fibrous is calcified and forms a phlebolith.
(3) The lumen of the vein may be re-established by gradual opening up of the interstices of the clot.
(4) The clot may disintegrate before it has firmly formed. If septic this produces pyæmia.
(5) The clot may be washed out *en masse* and form an embolus.

Effects on the Parts below the Thrombus.

Great swelling and solid œdema (*see* "Phlebitis.")

Enlargement of collateral veins.

EMBOLISM.

Definition.—A blocking of the blood-vessels by a substance carried thither by the blood-stream.

Regarded as to its effects, it is generally a disease of the arteries, but in its origin it is usually a disease of the veins.

Varieties of Emboli.

SIMPLE.

From the Heart: Vegetations from valves, or clots formed in dilated cavities.

From the Arteries: Atheromatous plates, clots from an aneurysm.

From the Veins: Thrombi.

AIR.—Introduced through a wound of a large vein, generally in the neck during inspiration. If large in amount it produces a frothy blood condition which clogs the heart and causes death.

FAT. A very rare condition after bone injuries; the minute vessels of the brain and lungs are chiefly affected.

INFECTIVE. Consist in infected portions of blood-clot and zooglœa masses of bacteria. Produce infarctions and abscesses of pyæmia.

MALIGNANT. Formed by the growth of soft, friable, malignant tissue, generally sarcoma, into the cavity of veins. Particles are washed away and cause metastatic growths, generally in the lungs.

PARASITIC. Ova and scolices of tænia, filaria, etc.

Pathological Results of Embolism.—These depend upon (1) The size and position of the vessel blocked; (2) The infective or simple character of the embolus; (3) The relation of the blocked vessel to collateral circulation.

IN SIMPLE EMBOLI affecting terminal arteries:—

Anæmia.

Necrosis (e.g., white softening in the brain).

Infarction: a cone of tissue whose vessels are all thrombosed, the embolus being at the apex. The infarct subsequently organizes into fibrous tissue and cicatrizes.

Hæmorrhagic Infarction occurs in very vascular organs, e.g., the lung, kidney, or spleen, and consists in a venous engorgement of the infarcted area.

IN INFECTIVE EMBOLI, infarction is followed by local suppuration forming *pyæmic abscesses*.

When the infected embolus lodges in an artery, an acute or inflammatory *aneurysm* may follow.

IN THE MAIN VESSELS OF THE LIMBS, gangrene may occur under conditions mentioned (*vide* "Gangrene").

Special Clinical Forms of Embolism.

CARDIAC AND PULMONARY EMBOLISM.—A large clot becomes entangled in the right side of the heart or in the pulmonary artery. Sudden death, accompanied by great pain over the heart, results.

IN THE BRAIN.—Generally the middle cerebral, supplying the motor cortex and motor tracts. Produces white or yellow softening and hemiplegia.

IN THE RETINA.—Permanent blindness.

Special Clinical Forms of Embolism, *continued.*

IN THE LUNG.—Sudden pain, dyspnœa, and hæmoptysis. Very rarely some local signs of pleurisy and consolidation. Simple infarcts cicatrize, infective form abscesses.

IN THE LIVER.—Usually infective, from the portal area, e.g., an appendicitis. Results in multiple abscesses.

IN THE SPLEEN OR KIDNEY.—Sudden pain in the affected organ, with occasional hæmaturia in the latter.

IN THE INTESTINE.—A large vessel is usually blocked. Tympanites, obstruction, and gangrene usually result.

PHLEBITIS.

Definition.—Inflammation of the vein wall.

Causes.

SIMPLE: Injury, pressure, gout, varicosity. Idiopathic cases are not uncommon.

INFLAMMATORY: Thrombosis, spreading from a septic focus. Inflammatory lesions outside the vein. Septic wounds.

Pathology.—In simple cases a plastic organizing thrombosis occurs. In septic cases varying degrees of acute inflammation attack (1) the inner coats, (2) the whole vein, (3) the tissues outside the vein. In the latter case an abscess is formed. Thrombosis usually spreads some distance up and down the vein.

Symptoms.—A hard, painful, cord-like swelling forms over the vein. Skin over this is dusky, congested, and œdematous. If the vein is superficial there are no other signs.

If it is the main deep vein of the limb, MASSIVE SOLID ŒDEMA occurs, with considerable lymphatic engorgement (white leg).

SUPERFICIAL VEINS ENLARGE in order to carry on the collateral circulation.

FEVER, with rigors, occurs, and is proportioned to the infectivity of the process.

ABSCESSES DEVELOP round an infective phlebitis.

Complications and Sequelæ.

Cardiac or pulmonary embolism follow the dislodgment of a thrombus.

Pyæmia results from disintegration of a septic thrombus.

Permanent œdema, with varicose veins, is left in the leg when the deep femoral is blocked.

Treatment.

Rest and elevation in bed for 6 weeks.

Belladonna applications for pain.

Excision of the veins in recurrent superficial phlebitis.

Incision, removal of clot with proximal ligature in infective phlebitis, e.g., in the jugular vein following acute mastoiditis.

VARICOSE VEINS.

Definition.—A redundance, tortuosity, lengthening, and thickening of a group of veins.

Distribution.

Saphena veins of the legs, veins of the rectum (hæmorrhoids), and of the testes (varicocele).

Causes.

Congenital over-development of the veins.

Absence or incompetence of the valves at the mouth of the saphenous veins.

Continued over-distention caused by standing.

Increased abdominal pressure, from pregnancy, corpulency, or tumour.

Blocking of the deep veins by thrombosis.

Anatomy.

THE INTERNAL SAPHENOUS VEIN is generally affected; the external more rarely.

The veins are dilated, thickened, lengthened, tortuous, and destitute of valves. A condition like atheroma frequently develops in their walls.

Localized pouches occur, or may constitute the main disease.

The tunica media is atrophied, the adventitia much thickened. In extreme instances the vessel gapes when cut across.

Signs.

The veins themselves form tortuous dilatations under the skin, chiefly on the inner side of the leg below the knee.

Congestion, œdema, eczema, and ulceration result from the impeded circulation in neglected cases. This is most marked above the ankle and in front of the tibia.

Pain, tiredness, and aching are usual symptoms.

Complications.

RUPTURE occurs from an injury of a thin pouch, or by ulceration. Bleeding may be severe.

PHLEBITIS is common and aggravates the congestive signs. Phleboliths may form in some of the veins.

EMBOLISM is comparatively rare.

Treatment.

PALLIATIVE.—Superficial support by elastic bandages or stockings, adjusted before the patient gets up.

Exercise, as distinguished from standing, is beneficial, as promoting the deep muscular circulation.

Unna's paste is useful for all congestive and inflammatory conditions.

Congestive ulcers must be treated as above described.

RADICAL OR OPERATIVE.—This is required (1) In candidates for military, police, and other public posts; (2) When congestive complications are present; (3) When great pain and aching exist.

Varicose Veins, Treatment, *continued.*

It is contra-indicated (1) When it is secondary to deep thrombosis coming on after, e.g., "white leg"; (2) When it is only marked during pregnancy; (3) When the condition is diffuse, painless, and well relieved by elastic bandages.

OPERATIONS.

TRENDELENBURG'S.—Consists in tying the saphenous vein just below the saphenous opening. It is indicated when (1) A marked impulse occurs in the veins on coughing, showing a valvular incompetence; (2) When pressure over the top of the vein prevents the varicose veins re-filling after they have been emptied; (3) When only the tributaries of the internal saphenous vein are involved.

MULTIPLE EXCISION or LIGATURE over all prominent veins is the most certain cure.

A judicious combination of the two methods gives the best results.

NÆVI AND OTHER VASCULAR TUMOURS.

Classification.—Tumours consisting entirely of blood-vessels are of four kinds: (1) Capillary nævi—birth marks, port-wine stains, etc.; (2) Venous or cavernous nævi—chiefly dilated veins; (3) Aneurysm by anastomosis—arteries and veins which open directly into one another; (4) Cirsoid aneurysm—purely arterial.

Capillary Nævi.—May be congenital, but usually develop soon after birth. Vary in size from a pin's head to half the body surface. Usually bright red or dusky red colour. May be flat or raised and nodular. When very large (e.g., half the body or over an entire limb) they are accompanied by hypertrophy of these parts. When raised and nodular they often ulcerate and bleed.

TREATMENT.—Excision. Electrolysis in some parts of the face. Painting with sodium ethylate or the actual cautery in very superficial nævi.

Cavernous Nævus.—Usually affects the subcutaneous tissues as well as the skin. The veins are often large cavernous spaces into which arteries open directly. A whole limb or one side of the body may be involved, and this is then generally hypertrophied.

TREATMENT.—This is only possible when the nævus is localized.

EXCISION is the ideal method for small tumours.

ELECTROLYSIS for the larger ones. A current of 200 milliampères is passed through the positive pole in the form of a series of needles embedded in the mass.

Aneurysm by Anastomosis.—A rare condition. It may be congenital, and is then associated with hypertrophy of the affected part. Masses of tortuous arteries and veins communicate freely with one another and produce a distinct bruit and thrill. It occurs in cancellous bones, or the cranial diploe, or in one of the limbs.

Treatment is the same as for cirsoid aneurysm.

Cirsoid Aneurysm. A mass of dilated varicose arteries. Occurs most frequently on the scalp or orbit, connected with the temporal, posterior auricular, and occipital or ophthalmic arteries, but may arise on the perineum, trunk, or limbs. A loud bruit is heard over it. The skin above and the bone beneath are atrophied by pressure. The hair falls out, and ulceration and hæmorrhage may result. Sometimes it spreads rapidly.

TREATMENT OF PLEXIFORM ANGEIOMATA (aneurysm by anastomosis and cirsoid aneurysm). Excision is usually out of the question. A series of ENCIRCLING LIGATURES or an attempt to ligature all the feeding vessels is the best method. Electrolysis is the only alternative.

DISEASES OF THE LYMPHATICS.

Acute Lymphangitis.—Occurs under the same conditions as cellulitis, into which it often merges. The inflamed lymph-vessels are seen as red and tender streaks running up to enlarged glands.

The treatment is that of the primary septic focus.

Chronic Lymphangitis may accompany syphilitic or tubercular diseases.

Division of the Thoracic Duct which opens into the junction of the left subclavian and jugular veins is followed by a chylous fistula.

This must be treated by LIGATURE, anastomotic channels carrying on the chyle circulation.

Lymphangiomata may be capillary or cavernous, and resemble nævi. They are often papillary, and the papillæ present clear vesicles. Their rupture gives rise to lymphorrhœa.

Cystic Hygromata, macroglossia, and macrocheilia consist of lymphatic dilatations of congenital origin (*vide infra*).

Chylous Ascites and chylous hydrocele are very rare conditions due to an obstruction to the thoracic duct by tumours or parasites.

Chronic Lymphatic Obstruction is due to (1) Tubercular or malignant disease in the glands and vessels, or (2) *Filaria sanguinis hominis.*

SIGNS of this are (1) Solid or lymphatic œdema; (2) Hyperplasia and connective tissue overgrowth; and (3) Lymphatic fistulæ with lymphorrhœa.

Elephantiasis Arabum is the result of infection by *filaria* introduced by mosquito bites. The adult worm is 3 inches long, and located in a lymph- or blood-vessel; the swarm of embryos block the lymph vessels and cause the obstruction. The external genitals and legs are chiefly affected, and form huge warty, hypertrophied masses.

Partial or complete AMPUTATION or the ligation of the main arteries are the modes of treatment.

AFFECTIONS OF THE LYMPH-GLANDS.

Acute Lymphadenitis is always secondary to some septic lesion in the associated skin areas.

ANATOMY. The glands become swollen and matted together, and periadenitis quickly follows, producing a diffuse inflammatory swelling, which soon forms an abscess.

SITUATION.

In the Submaxillary Triangle of the neck it is secondary to buccal, tonsillar, and dental diseases.

In the Posterior Triangle, to pediculosis or scalp ulcers or wounds.

In the Axilla, to sepsis in the arm or breast.

In the Groin, to sepsis of the genitals, abdominal wall, perineum, and anus (this generally affects the oblique set of glands parallel to Poupart's ligament); to sepsis of the leg, especially on the inner side.

In the Ham, to disease on the outer side of the leg and foot.

TREATMENT is by fomentations and free incisions.

CHRONIC LYMPHADENITIS may be simple, syphilitic, or tuberculous.

Tuberculous Lymphadenitis.

ETIOLOGY. Children or young adults. Bad food and hygiene. Frequently there is some predisposing cause for enlargement, e.g., carious teeth, tonsillitis, or pediculosis.

DISTRIBUTION.—The submaxillary, carotid, and supraclavicular glands are most often affected, and in this order; the axillary and inguinal glands much more rarely, owing to much fewer predisposing causes for enlargement or points of entry for the tubercle bacillus.

PATHOLOGY. The glands go through the following stages :—

(1) Enlargement, with tuberculous infiltration.
(2) Caseation, sometimes followed by calcification or cicatrization.
(3) Suppuration, which may be of a tuberculous or mixed type.
(4) Periadenitis always occurs before long, and serves to mat together neighbouring masses of glands and fixes the glands to surrounding tissues.

SIGNS. Three stages are usually noted :—

(1) Simple enlargement, without much tenderness, in which individual glands can be distinguished.
(2) The stage of periadenitis, in which the glands become fused together and more firmly fixed in their surrounds.
(3) The stage of suppuration, when an abscess (usually cold) develops. Chronic sinuses are left by the bursting of the abscesses, and if these heal they leave scars which are puckered, keloidal, and vascular.

TREATMENT.

PALLIATIVE, by high feeding, fresh air and removal of all local sources of irritation.

OPERATIVE. This is contra-indicated when active lung disease co-exists or when the opsonic index is low. The glands must be dissected out or abscesses opened and scraped.

In the neck. The incisions should be made (1) below and parallel to the jaw, (2) behind the sternomastoid, so as to leave inconspicuous scars. In bad cases it may be necessary to divide the sternomastoid or dissect out the internal jugular vein.

DIAGNOSIS.

SIMPLE CHRONIC ADENITIS affects only one or two glands, and is stationary.

SEPTIC ADENITIS has an acute course, with signs of inflammation.

HODGKIN'S DISEASE affects many groups of glands simultaneously; the glands remain discrete longer, and do not break down. Marked leucocytosis is present.

Other lymph-gland tumours (*vide infra*).

Other Varieties of Lymph-Gland Enlargement.

LYMPHADENOMA.—Simple enlargement and overgrowth of one or more glands, which do not become matted or inflamed, and do not suppurate.

TREATMENT by arsenic produces great diminution of swelling. Early removal is called for.

HODGKIN'S DISEASE—Is a lymphadenoma affecting many groups of glands simultaneously. It is associated with leucocytosis.

LEUKÆMIA.—Causes swelling of glands, bones, and spleen, with characteristic blood changes.

LYMPHOSARCOMA.—Is a rare primary disease in the glands. It occurs in the neck and mediastinum. The growth is very rapid, vascular, and fatal.

SECONDARY MALIGNANT DISEASE—Is common.—Epithelioma, scirrhus, or sarcoma may arise in the neck, axilla, or groin.

CHAPTER XIV.

INJURIES AND DISEASES OF NERVES.

INJURIES OF NERVES.*

Causes.

TRACTION DURING BIRTH.
CONTUSIONS AND STRAINS.
DISLOCATIONS AND FRACTURES.
} Producing rupture of some or all the nerve fibres.

PRESSURE: Growth of tumours—Aneurysm—Displaced bones (Fracture, dislocation, Cervical rib)—Inclusion in callus—Pressure of crutches or splints—Inflammatory products in bony canals.

WOUNDS.

Effects of Total Division of a Mixed Nerve.

IMMEDIATE.—Paralysis of muscles. Anæsthesia of skin: only partial, owing to the overlapping of sensory areas. Vasomotor paralysis, producing first hyperæmia, then anæmia of parts. Glands do not respond to ordinary stimuli.

LATE EFFECTS OF NERVE DIVISION:—

CHANGES IN THE NERVE.—Retraction of divided ends.

Formation of a hæmatoma, especially at the proximal end.

Organization of the hæmatoma into a bulbous nerve-end (traumatic neuroma). This contains bundles of new nerve fibrillæ embedded in fibrous tissue.

The peripheral end shrinks and atrophies.

Wallerian degeneration occurs below the injury, and above as far as the first Ranvier's node. Begins at the fourth day after injury. Medullary substance breaks up into fat globules. Axis cylinders disappear in one month. Leucocytes invade and replace the nerve fibres.

CHANGES IN MUSCLES.—Muscle cells atrophy, or are replaced by fibrous tissue and fat. Deformities occur from unopposed action of the unparalyzed muscles.

ELECTRICAL REACTIONS.—Excitability by faradic current is rapidly lost. Excitability by galvanic current is at first increased, and only lost very slowly. The response to galvanism is a peculiar sluggish contraction called "the reaction of degeneration." A greater contraction occurs at the anode than at the kathode on closing the current. As long as any response to electrical stimuli remains there is hope of repair.

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* Sherren, "On Injuries of Nerves."

SENSORY CHANGES.—Peripheral sensory nerves are of three kinds physiologically.

1. NERVES OF DEEP SENSATION.—These perceive deep pressure and pain, and the movements and position of bones and joints. They probably run in the muscles, tendons, and bones, and their functions are seldom if ever lost by peripheral nerve division.
2. PROTOPATHIC NERVES.—These respond to painful skin impressions, e.g., a prick, and distinguish extreme temperatures. Their area of distribution is badly localized, and stimulaton of them gives a widely radiating, tingling feeling. They are concerned with the production of reflex movements.
3. EPICRITIC NERVES.—These distinguish light touches, e.g., by a hair or brush, and also small differences of temperature. They convey a well-localized sensation.

IN DIVISION OF A SENSORY NERVE: (1) are unaffected; (2) are affected over a small and variable extent, as there is much overlapping of adjacent protopathic nerves; (3) are affected over a constant and well-defined area, considerably larger than that of (2).

IN DIVISION OF A POSTERIOR ROOT there is always a larger loss of protopathic sensation than of epicritic. Thus the peripheral nerve is the unit of epicritic nerve supply, and the posterior root is the unit of protopathic nerve supply.

CHANGES IN THE JOINTS.—A plastic synovitis occurs, resulting in ankylosis. Especially noticed in the small joints of the fingers.

TROPHIC CHANGES.

(*a*) IN COMPLETE DIVISION of the nerve without irritation or neuritis. Skin becomes rough, scaly, and œdematous. Glands atrophy, and hence the skin is very dry. Atrophy of the bones or cessation of growth occurs. Hair and nails break, or fall off.

(*b*) WHEN THE DIVIDED NERVE IS IRRITATED (the commonest condition). Skin is thin, shiny, bluish red—"glossy skin." Vesicular and pustular eruptions occur. Chilblains are common. Excessive sweat secretion. Ulcers and whitlows. Hair falls out or breaks off. Nails are brittle and ridged, or lost by perionychia.

IN BOTH CASES.—Temperature falls as much as 8° F. in affected parts.

CHANGES IN THE CENTRAL NERVOUS SYSTEM.—Spasms of a reflex nature; epileptic fits and dementia (both very rare).

RECOVERY OF A DIVIDED NERVE.—Can only occur when the two ends are brought close together, an inch separation being probably a maximum. The bulbous nerve end offers great obstacles to repair.

New axis cylinders: (*a*) Grow down from proximal to distal part; (*b*) Develop in the distal segment.

Recovery of a Divided Nerve, *continued.*

Protopathic sensation returns first, and with it the tendency to trophic changes (blisters, etc.) disappears. This takes place in periods varying from six weeks to one year.

Epicritic sensation is the last function to be recovered, and the recovery is seldom, if ever, complete. It returns in six months to two years or more.

The time at which sensory recovery begins does not vary with the position of the lesion, but the time occupied by the recovery varies directly with the distance between the point of section and the periphery.

Motor recovery takes place first in the muscles nearest to the point of section, and eventually is more complete than the sensory recovery.

Effects of Partial Division of a Mixed Nerve.

MOTOR CHANGES.—Instead of the typical reaction of degeneration, there is with failure of reaction to the faradic current a ready and brisk reaction to galvanism, with no polar reversal.

SENSORY CHANGES.—Loss of epicritic sensation is the most marked and most constant sign.

A considerable portion of a mixed nerve can be divided up to about one-third of all its fibres without producing any appreciable effect.

Treatment.

IF COMPLETE DIVISION is diagnosed,

OPERATE immediately on all important nerves. Refresh the ends, and sew together. Prevent tension by tension sutures, and also by the position of the part.

SECONDARY SUTURE may be undertaken up to three years from the injury.

IF ONLY PRESSURE or partial laceration exist,

Wait to see whether natural recovery will take place.

The Diagnosis of Nerve Division has to be made from :—

SPINAL CORD INJURY.—In this there is total loss of sensibility to either pain, heat, cold, tactile sensation, or deep sensation over a given area opposite to the side of the lesion, combined with a loss of movement and the sensation of passive movement on the same side as the lesion.

HYSTERIA.—In this there is a complete loss of all forms of sensation over an area the upper margin of which is a simple circle, and this constitutes the so-called "stocking anæsthesia." With this there may be a flaccid paralysis, but the electrical reactions are quite unchanged.

ISCHÆMIC PARALYSIS.—In this there may be areas of sensory loss with trophic changes, but the characteristic shortening of the flexor muscles without actual paralysis is the clue to the condition.

NEURALGIA.

Definition.—Pain in the area of a nerve distribution without any primary nerve lesion. The pain often recurs daily at the same time.

Varieties.

A. SYMPTOMATIC OR SECONDARY.

TOXIC, e.g., malarial, gouty, lead.

REFLEX, e.g., neuralgia secondary to carious tooth or glaucoma.

PRESSURE, e.g., of aneurysm, or new growth on nerve roots.

B. IDIOPATHIC OR PRIMARY.—Commonest in the fifth nerve. Also in Intercostals—Breast—Ovaries—Testes—Joints.

IDIOPATHIC NEURALGIA,[*]

With especial reference to Trigeminal Neuralgia.

Etiology.—Cause unknown. An ascending neuritis with arteriosclerosis has been suggested, but not demonstrated. Women more often than men. Generally after 40.

Symptoms.

PAIN.—Paroxysmal attacks (epileptiform). The second or third division of the nerve is alone affected at first, but soon the attacks involve the other two divisions. First escapes longest. Each attack lasts only a few seconds to a minute. Pain during the attack is excruciating, sharp, and lancinating. Attacks at first are seldom, but recur more frequently. Attacks are often brought on by sensory stimuli, e.g., cold draught, or brushing the hair. Constant aching pain follows the paroxysm, and may last until the next occurs.

SENSORY AND TROPHIC CHANGES.—Pressure over nerve trunks is very painful. Skin tender, and at places hyperæmic and œdematous. Profuse sweating, with lachrymation and increase of nasal discharge. Skin becomes smooth and shiny, and hairs fall out.

MOTOR SYMPTOMS.—Associated muscles thrown into reflex spasms or twitchings.

Treatment of Idiopathic Trigeminal Neuralgia.

DRUGS.—Salicylates, quinine, iodides, croton chloride. All lose their effects rapidly.

Avoid morphia at all costs.

OPERATIVE.

(1) NERVE STRETCHING AND DISTAL NEURECTOMY give such temporary relief that they are not used now.

Lingual neurectomy is often performed for cancer of tongue.
Inferior dental nerve may be removed for toothache.

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[*] J. Hutchinson, jun., "Trigeminal Neuralgia."

Operative Treatment of Idiopathic Trigeminal Neuralgia, *continued.*

(2) PROXIMAL NEURECTOMY.—Nerves are removed at their exit from the skull. The zygoma and part of the ramus of the jaw are removed, and the second and third divisions removed as near the skull as possible, together with Meckel's ganglion.

Very difficult operation. Apt to be incomplete, and therefore is uncertain in its results. Produces adhesions round temporomaxillary joint.

(3) EXCISION OF GASSERIAN GANGLION. This is the only method so far which has given permanent results.

PTERYGOID ROUTE.—Zygoma and coronoid process turned aside—Second and third divisions found—Base of skull trephined outside foramen ovale—Ganglion torn out piecemeal.

TEMPORAL ROUTE. (Hartley Krause operation).—Horseshoe flap down to the bone, with base at zygoma. Bone turned back in osteoplastic flap, or removed by trephine for 1½ inch circle. Dura mater pushed back. Middle meningeal artery tied or plugged. Ganglion removed after cutting second and third divisions. Eyelids ought to be sewn together for ten days to protect from irritation, otherwise corneal ulcer and blindness may follow.

This is the method of choice in all severe cases.

(4) DIVISION OF THE ROOT OF THE GASSERIAN GANGLION through the posterior cranial fossa.

(5) INJECTION OF THE BRANCHES OF THE NERVE with osmic acid or absolute alcohol.

(6) REMOVAL OF SUPERIOR CERVICAL SYMPATHETIC GANGLION.

AFFECTIONS OF SPECIAL NERVES.

Optic Nerve.

RUPTURED by fracture through anterior cranial fossa.

COMPRESSED or INFLAMED by intracranial diseases, especially meningitis or tumours.

INJURED by orbital growths, hæmorrhage, or cellulitis.

SYMPTOMS.—Blindness, optic atrophy, or optic neuritis.

Third Nerve.

INJURED by fractures through sphenoidal fissure—Orbital tumours or aneurysms.

DISEASED by syphilitic disease of brain.

SYMPTOMS.—Ptosis, or drooping of upper eyelid—External squint—Diplopia when looking inwards—Dilated pupil—Loss of accommodation—Slight exophthalmos.

Fourth Nerve.—Injuries and diseases as in the case of the third.

SYMPTOMS.—Squint and diplopia when looking downwards.

Sixth Nerve.—Injuries and diseases as in the third.

SYMPTOMS.—Internal squint—Diplopia when looking outwards.

INJURIES AND DISEASES OF THE SPHENOIDAL FISSURE OR CAVERNOUS SINUS

PRODUCE: Paralysis of all the muscles of the eye—Anæsthesia of the cornea and forehead—Venous congestion of the eye and conjunctiva.

OPHTHALMOPLEGIA EXTERNA, i.e., paralysis of all the eye muscles: Without congestion—Caused by syphilitic or tabetic disease of the floor of the third ventricle.

(For diseases of the fifth nerve see "Trigeminal Neuralgia.")

Seventh, or Facial Nerve, or its centres, may be diseased or injured anywhere between the cerebral cortex and the branches of distribution.

INTRACRANIAL LESIONS.

CEREBRAL CORTEX.—Injury—Pressure—Hæmorrhage. Part of the opposite side of the face paralyzed.

CORONA RADIATA.—Hæmorrhage or thrombosis. Lower half of opposite side of face paralyzed. Eyelids and occipitofrontalis escape. Combined with paralysis of arm, and perhaps the leg.

IN PONS.—Hæmorrhage or growth. Paralysis, with atrophy of the same side of the face from affection of the facial nuclei. Paralysis of the opposite side of the body from affection of the pyramidal tract of the opposite side above the decussation of the pyramids.

NERVE ROOT between the brain and the bone.—By injuries or tumours. Paralysis of the whole of the same side of the face. Generally associated with nerve deafness.

CRANIAL LESIONS.

FRACTURE OF THE BASE OF THE SKULL running through the internal auditory meatus. Causing immediate laceration of the nerve, or later implication in callus.

OTITIS MEDIA causing compression of the nerve in the aqueductus fallopii.

INJURY OF NERVE during mastoid operations.

A loss of taste in the anterior two-thirds of the side of the tongue follows a division of the nerve between the geniculate ganglion and the joint where the chorda tympani leaves the trunk.

EXTRACRANIAL LESIONS.—Injury of the nerve by operations—Cold or inflammation—Tumours (especially malignant) of the parotid.

Produce total facial paralysis.

FACIAL PARALYSIS.—Eyelids cannot be closed. Eyeball is rolled up on attempting to shut the eye. Corneal ulceration sometimes results from exposure. Epiphora from want of apposition of lower eyelid to eye. Face is immobile and the wrinkles smoothed out. Face is drawn to the opposite side. Cheek is flabby, and food collects between cheek and teeth from paralysis of the buccinator.

Paralysis of Seventh, or Facial Nerve, *continued.*

Prognosis.—In cerebral hæmorrhage—recovery generally takes place. In pontine hæmorrhage—atrophy occurs and recovery seldom (patient usually dies). In intra-osseous lesions—slow recovery is the rule. When the trunk of the nerve is divided, paralysis may be permanent.

Treatment.—Expectant in most cases. When paralysis is permanent: Grafting the hypoglossal or part of the spinal accessory nerves into the trunk of the facial.

FACIAL TIC OR HISTRIONIC SPASM.—Caused by some obscure central irritation. Results in involuntary clonic spasms of one side of the face.

Treatment.—Stretching the facial nerve.

Auditory Nerve and its Centres.—Tumour or injury of the opposite temporosphenoidal lobe, fracture of the base of the skull through internal meatus, produce incurable deafness.

Vagus.

TRUNK is injured by:—Fractures through the jugular foramen—Operations or tumours in the neck.

Symptoms: Vomiting—Inhibition of heart's action—Palpitation—Laryngeal paralysis.

THE RECURRENT LARYNGEAL NERVE is injured by subclavian aneurysm on the right side—Aortic aneurysm on the left side—On both sides by injuries, operations, or malignant growths in the neck.

Produce laryngeal paralysis—especially abductor paralysis. Hoarse voice. Asphyxia if bilateral.

Eleventh or Spinal Accessory Nerve.

INJURED OR DIVIDED BY

(*a*) Fractures through the jugular foramen.

Produce paralysis of larynx and pharynx through affection of accessory portions. Paralysis of sternomastoid and trapezius (partial).

(*b*) Operations in the neck, especially those for the removal of glands.

Produce paralysis of sternomastoid (slight), and more complete of trapezius, with wasting and drooping of shoulder.

SPASMODIC WRY NECK.—Clonic contraction of the sternomastoid and the small rotator muscles of the head.

Caused by central irritation of an obscure nature affecting spinal accessory and upper cervical nerves.

Treatment.—Excision of part of the spinal accessory, together with posterior divisions of the upper three cervical nerves.

Hypoglossal Nerve.—Injured by wound or operation in neck, or by carotid aneurysm or tumour. Produces unilateral paralysis and hemiatrophy of tongue. The tongue when protruded deviates to the opposite side of the mouth.

Sympathetic Nerve in the Neck—May be involved by tumours, or injured by stabs. Irritation produces dilatation of the pupil and palpebral fissure, with exophthalmos. Also unilateral sweating.

Paralysis produces contraction of the pupil, narrowing of the palpebral fissure, and retraction of the eye, with dryness of the skin.

Phrenic Nerve.—Arises from the fourth cervical and goes to the diaphragm. Wounds in the neck may divide it, but unilateral division causes no symptoms. Irritation of the nerve is said to cause cough or hiccough.

Brachial Plexus.

CAUSES OF INJURY.

SUPRACLAVICULAR.—(*a*) Indirect violence. Excessive or sudden traction on the arm in attempting to save a fall, or at birth. (*b*) Direct injury. Cervical rib, fractured clavicle, or stab wounds. Usually affects the first dorsal.

INFRACLAVICULAR.—The various incidents during and after dislocation of the shoulder joint.

FUNCTIONS OF THE BRACHIAL ROOTS.

THE POSTERIOR ROOTS.—Of these only two have well-defined areas of distribution, viz., the fifth cervical and the first dorsal, lesions of which cause a preaxial and postaxial anæsthesia of the arm respectively.

THE ANTERIOR ROOTS.

Fifth cervical—the deltoid, rotators, biceps, coracobrachialis, brachialis anticus, supinators, and rhomboids.

Sixth cervical—pronators, radial extensors, clavicular part of pectoralis major, serratus magnus.

Seventh cervical—triceps, ulnar extensors, finger extensors, lower part of pectoralis major.

Eighth cervical—flexors of wrist and fingers.

First dorsal—Intrinsic muscles of the hand.

There are three common types of brachial plexus injury :—

1. WHOLE PLEXUS.—All the muscles of the arm are paralyzed except the rhomboids and serratus. There are usually some signs of injury of the cervical sympathetic. In SUPRACLAVICULAR rupture sensation is lost over the whole arm except on the inner side adjacent to the axilla, which is supplied by the intercosto-humeral nerve. In INFRACLAVICULAR rupture the loss of sensation is complete.

2. UPPER ARM OR ERB-DUCHENNE TYPE.—Essentially a rupture of the fifth cervical anterior primary division. It is commonly caused by traction on the arm in a direction towards the feet. Paralysis of the deltoid, rotators, biceps, brachialis anticus, and supinators. The arm lies at the side, and the forearm is pronated. There is no loss of sensation. Occasionally the deltoid and rotators are paralyzed alone from a partial rupture of the fifth cervical in its upper part.

Brachial Plexus, *continued.*

3. LOWER ARM OR KLUMPKE TYPE.—Is caused by a rupture of the first dorsal nerve. Caused by traction on the arm upwards. Paralysis of the intrinsic muscles of the hand, with sympathetic paralysis. Sensation is lost over the inner side of the arm and forearm.

THE INNER CORD OF THE PLEXUS may be injured by a subcoracoid dislocation. The symptoms are those of injury of the ulnar nerve, together with the small hand muscles supplied by the median (inner head of the median).

THE OUTER CORD OF THE PLEXUS is occasionally injured in dislocations. There will be paralysis of the biceps, coracobrachialis, and all the muscles supplied by the median, except those in the hand.

THE POSTERIOR CORD is the most rarely injured part of the infraclavicular plexus. The symptoms will be those of lesions of the musculospiral and circumflex nerves.

Posterior Thoracic, or Nerve of Bell, which arises from the fifth, sixth, and seventh cervical nerves, and goes to the serratus magnus, is often paralyzed by itself as the result of injury or neuritis.

SIGNS.—Winged scapula. Inability to raise the arm above a right angle.

The Circumflex Nerve may be injured by blows, fractures of the surgical neck of the humerus, or by dislocations.

SIGNS.—Paralysis and wasting of the deltoid and teres minor.

The Musculospiral is the most commonly injured of all the spinal nerves.

CAUSES.—Fractures and dislocations of the humerus and shoulder. Pressure of crutches at the posterior axillary fold. Pressure on the arm under the body during anæsthesia, or a drunkard's sleep.

SYMPTOMS.—Paralysis and wasting of the triceps, supinators, and extensors of the thumb and fingers and wrist.

Wrist-drop is the most prominent sign, the hand being held in a position of pronation. The fingers and thumb cannot be properly extended, but the terminal phalanges of the fingers can be extended by the interossei.

If the lesion is in the upper third of the arm (rare), there will be sensory loss over the back of the radial side of the hand and thumb. If the lesion is in the lower third of the arm (common type), there is no sensory loss, owing to the fact that the external cutaneous branches of the musculospiral anastomose with the external cutaneous nerve, which in its turn anastomoses with the radial nerve. In the same way division of the radial nerve high up in the forearm produces no sensory loss.

Median Nerve.—Usually injured by wounds above the wrist.

SYMPTOMS.—Paralysis and wasting of three short thumb muscles (the abductor, opponens, and part of the flexor brevis), with defective opposition of the thumb movement, also of the outer two lumbricals, which, if the interossei are intact, give no sign.

The paralysis of the opponens pollicis is manifested by inability to bring the thumb across the hand parallel to the palmar surface. It is difficult to detect, because the flexors and adductors together may simulate this movement.

ANÆSTHESIA (epicritic loss) of the palmar aspect of the thumb and adjacent two-and-a-half fingers, and of the dorsal aspect of the last, or last two phalanges of the same. Trophic changes will be evident.

If the nerve is injured at the elbow or in the upper arm: Paralysis of both pronators, with loss of pronation, and of all the flexors, except part of the flexor profundus and the flexor ulnaris.

Ulnar Nerve.

CAUSES OF INJURY.—Wounds or dislocations, especially in the neighbourhood of the elbow.

SYMPTOMS.—Paralysis of the lumbricals (inner two) and all the interossei, causing the *main-en-griffe*, i.e., hyperextension of the metacarpo-phalangeal joints and flexion of the two interphalangeal joints. Wasting seen between the metacarpals.

Paralysis of the adductor muscles of the thumb, and all the short muscles of the little finger, with wasting at the thenar and hypothenar eminences.

The paralysis of the adductors is shown by inability to bring the thumb against the index finger in a direction at right angles to the palmar surface. The long flexor or extensor may simulate this movement.

Paralysis of the flexor carpi ulnaris and part of the flexor profundus, with weakened hand-grasp and tendency to radial abduction of the hand.

ANÆSTHESIA (epicritic loss) of the little and half the ring fingers on both sides. Also of the ulnar part of the hand.

If divided just above the wrist, only the hand paralysis is seen, and there is no dorsal anæsthesia.

In all cases deep sensibility in the hand is only lost when the nerve is divided high up before it goes to the muscles, or when the flexor tendons as well as the terminal nerves are divided.

The Cauda Equina may be injured by falls and blows apart from or together with injuries of the spine. The sensory loss is of the root type, i.e., the protopathic is larger than the epicritic loss, usually being limited to a saddle-shaped area on the buttocks.

The Cauda Equina, *continued.*

DISTRIBUTION OF THE NERVES OF THE SACRAL PLEXUS.

Fourth lumbar—the posterior muscles of the leg below the knee and the tibialis anticus.

Fifth lumbar—the anterior leg muscles (except the tibialis anticus) and the peronei.

Second sacral—the glutei and hamstrings.

Third and fourth sacral—the levator ani, sphincter ani, and perineum.

The Sciatic Nerve—Is very seldom injured, owing to its deep position and great strength. The signs are those of the internal and external popliteal nerve injury, combined with paralysis of the hamstrings.

The external division (external popliteal) is often injured, whilst the internal escapes. In lesions of the whole nerve there is a very limited sensory loss on the inner side of the leg.

SCIATICA is neuralgia of the sciatic nerve.

CAUSES.—(*a*) Doubtful nature, e.g., cold, neuritis, rheumatism, gout, syphilis; (*b*) Pressure on the nerve, inside or outside the pelvis, by abscesses, aneurysm, or new growths; (*c*) Pressure on the nerve roots by caries, new growth, or injuries of the spinal column; (*d*) Chronic spinal cord diseases, e.g., tabes.

SYMPTOMS.—Pain down the back of the thigh, increased by pressure on the nerve, or by flexing the hip-joint when the knee is straight (stretches the nerve).

TREATMENT.—Drugs suitable for rheumatism, neuritis, etc. Absolute immobility for several weeks by bandaging the limb to a long Liston's splint. Nerve stretching.

External Popliteal Nerve.—May be torn or injured as it winds round the biceps tendon and neck of the fibula.

ANÆSTHESIA of the dorsum of the foot and PARALYSIS of the extensor and peroneal muscle groups. TALIPES EQUINO VARUS results.

Internal Popliteal Nerve—Is seldom injured. Paralysis of the calf and flexor muscles of the toes, with some anæsthesia of the sole of the foot, and TALIPES CALCANEO-VALGUS.

Anterior Crural Nerve—Is seldom affected. Its injury causes paralysis of the quadriceps extensor muscle and sartorius, with flexion of the knee and anæsthesia of the greater part of the thigh and leg.

CHAPTER XV.

AFFECTIONS OF THE SKIN.

Carbuncle.—A patch of infective gangrene affecting the subcutaneous tissues.

CAUSES.—Infection with *Staphylococcus pyogenes aureus*. Albuminuria, or diabetes are common antecedents. Local abrasions or contusions sometimes precede.

PATHOLOGY.—Acute inflammatory exudation is caused by the cocci. The dense fibrous tissue under the skin does not yield, and the compression strangles the vessels. Septic gangrene results from these causes. Later, the living tissues suppurate round the septic slough and form pus and granulations.

Hence, the following zones are distinguished from within outwards: (1) Central slough or core; (2) Layer of pus; (3) Layer of granulations; (4) Inflamed tissues.

Then the pus breaks through the overlying skin in one or two places and the slough is discharged, the cavity being filled up by granulation and cicatrization.

SIGNS.—Localized, red, brawny, painful, swollen area of skin, from one to six inches in diameter. There may be slight enlargement of lymph-glands. In one to two weeks the skin becomes vesiculated, and then shows several grey points. These burst, and pus oozes out. The openings run together, and some time later the slough comes away, the suppuration becoming freer all the time. The cavity left heals by granulation.

Extension may occur, especially if septic poultices are used, by infection of neighbouring hair follicles.

DISTRIBUTION.—Nape of neck, back, nates, and face, are the commonest sites. Usually single.

COMPLICATIONS.—Septic thrombosis of the sinuses may occur with carbuncle of the face. Septicæmia, pyæmia, and acid intoxication with coma—especially in diabetes.

SYMPTOMS.—General malaise, without much rise of temperature. If general septic infection occurs it will cause the usual symptoms.

DIAGNOSIS.—BOILS are multiple, conical, and have only one opening.

GUMMATA are painless, with but little inflammation. The discharge is gummy, the edges clean cut and sharply defined, the slough like wet wash-leather.

ANTHRAX.—A black slough surrounded by a ring of vesicles. Very marked œdema and swelling of lymph-glands. Little local pain, but marked rise of temperature.

Carbuncle, *continued.*

PROGNOSIS is good, unless albuminuria or diabetes exist, or sinus thrombosis or general septic infection occur.

TREATMENT.—Generous diet, with tonics or stimulants. Free opening and scraping away the sloughs, applying pure carbolic. If this is refused, fomentations should be antiseptic to prevent fresh foci of infection, and Bier's cupping-glasses will hasten the separation of sloughs.

A Furuncle or Boil is essentially the same as a carbuncle. A boil differs from a carbuncle in the following :—It affects the skin only, and not the subcutaneous tissue. It opens by a single opening, instead of several. It is multiple very frequently. Usually occurs from the infection of a hair follicle or sweat gland.

FURUNCULOSIS is a disposition to repeated crops of boils.

TREATMENT.—LOCAL AND PROPHYLACTIC.—Lotions of spirit and biniodide, 1–1000.

GENERAL.—Injection of staphylococcal vaccines under opsonic control.

Corn.—A hard keratinous mass of epidermis with a deep-growing conical centre, which presses on the tender parts of the deep skin.

CAUSES.—Abnormal pressure—badly fitting boots, projecting bony processes.

LOCALITY.—Toes, especially little and great, and over the prominences of talipes.

VARIETIES.—Hard or soft, according to whether they are exposed to the moisture of sweat or not.

TREATMENT.—Excision. Salicylic acid ointment or lotions. Pads to relieve pressure.

Wart.—Papilloma of the skin. Often multiple, and then usually on the hands of children. They appear sometimes quickly in crops, and disappear equally quickly. Possibly they are locally infective, i.e., they disseminate themselves.

TREATMENT.—Soften with salicylic ointment, and then touch with pure acetic or nitric acids.

VENEREAL WARTS are very large villous masses, which occur on the external genitals, glans, prepuce, or labia, as the result of the irritation of discharges.

TREATMENT.—Excision, with cautery of the base ; or application of calomel or acid nitrate of mercury.

Lupus Vulgaris.—A very chronic tuberculous disease of the skin.

OCCURS in children, or adults under 30. Attacks the nose, cheeks, lips, eyelids, nasal, oral, and lachrymal mucous membranes, and ears. Less often the fingers, toes, or trunk.

SIGNS.—Nodules appear deep in the skin, and then run together. Each nodule is red and raised, and resembles "apple jelly." The skin becomes infiltrated and ulcerated, fresh nodules appearing on the spreading margin. One edge may heal whilst the other extends. The ulcer is covered with granulations, and over these thick scabs form by the drying of the purulent secretion. Deep tissues, e.g., the cartilages of the nose or ears, are attacked and destroyed. The scars left are puckered, thin, and vascular.

COURSE.—The disease runs a course of many years, but occasionally dies out spontaneously.

COMPLICATIONS.—Epithelioma may form in the scar. Other tuberculous diseases very rarely arise.

DIAGNOSIS depends on the tuberculous nodules, the slow superficial ulceration, the congested scar tissue.

SYPHILITIC ULCERATION is much more rapid and deep. Other signs are usually present. The scars are thin, pale, and supple.

LUPUS ERYTHEMATOSUS.—(*Vide infra*).

TREATMENT.—Removal by scraping, and the subsequent application of caustics, e.g., zinc chloride.

Finsen light or X rays.

Vaccine treatment by a vaccine composed of tuberculin, and usually another vaccine prepared from the patient to combat the septic infection by staphylococci.

Lupus Erythematosus.

OCCURS generally in adult women, on the face in symmetrical patches, like a butterfly, the wings on the cheeks and body on the nose.

SIGNS.—Smooth hyperæmic patch, covered with a fine branny desquamation. Associated with some seborrhœa. It does not ulcerate, but leaves a thin white scar surface in the middle, whilst the edge spreads.

TREATMENT.—Tar and mercury ointments. Light treatment.

AFFECTIONS OF THE NAILS.

Onychia, or Peri-onychia.—An inflammation of the nail matrix.

SEPTIC ONYCHIA is an ungual whitlow caused by a septic wound. Pus collects under the nail.

TREATMENT.—Removal of the nail and scraping the granulations.

SYPHILITIC ONYCHIA occurs in congenital and acquired syphilis in the secondary stage. It yields to mercury.

Ingrowing Toe-nail.—Usually affects the great toe.

Caused by boot pressure, the edge of the nail being pressed into the soft tissues and causing a chronic ulceration.

TREATMENT.—Removal of nail and thorough scraping of the matrix.

Onychogryphosis.—A deformed and overgrown great toe-nail seen in old people. When it becomes painful it should be removed.

AFFECTIONS OF THE SEBACEOUS GLANDS.

Adenoma Sebaceum, or Lipoma Nasi.—Forms a bulbous hypertrophied mass on the skin of the nose (*vide* section on nose diseases.)

Carcinoma Sebaceum causes a rodent ulcer (*vide* p. 59).

TREATMENT.—(1) Excision of the whole thickness of the affected skin and subcutaneous tissue, with half-an-inch margin of healthy tissue; subsequent skin grafting. (2) Repeated exposure to the X rays. This is suitable for inoperable cases, and those where removal would cause great deformity, or when operation is refused.

Sebaceous Cysts.

OCCUR on any part of the body, but especially on the hairy parts, on the scalp behind the ears, or in the eyelids.

SIGNS.—A fluctuating swelling in the skin which moves freely over subjacent tissues. A blocked mouth of a sebaceous gland is often seen.

ANATOMY.—The cyst wall may be epithelial, or from degenerative changes calcareous (atheromatous), or it may present adenomatous growth. The contents are pultaceous, and consist of fat, epithelial débris, and cholesterin. They may form a calcareous mass.

COMPLICATIONS.

A SEBACEOUS HORN may grow up by superposition of successive layers of epithelium.

A FUNGATING ADENOMA may follow its rupture.

DIAGNOSIS.

DERMOID CYSTS occur in special situations and always lie deep to the skin.

LIPOMATA are below the skin and are lobulated.

ABSCESSES have a more rapid course and affect the deeper tissues.

TREATMENT.—Dissect out.

Molluscum Contagiosum.—Yellowish-white nodules about the size of a pea.

OCCUR on the face, or less often other parts of the skin.

CONSIST of a central core of epithelial cells supported by a fibrous stroma. Bodies like psorosperms are found, but are the result of cell degeneration.

They are locally contagious.

TREATMENT by excision.

CHAPTER XVI.

AFFECTIONS OF MUSCLES, TENDONS, SYNOVIAL SHEATHS, AND BURSÆ.

Traumatic Affections of Muscles.

RUPTURE OF MUSCLE SHEATH.—Especially in the biceps cubiti and rectus femoris. Causes a soft hernia-like protrusion of muscle fibre when the latter contracts.

Rarely necessary to operate.

DISLOCATION OF TENDONS.—Most common in the neck, long tendon of biceps, peroneus longus. Great pain and stiffness after some sudden exertion.

TREATMENT is immobilization in plaster for 3 to 6 weeks in a position of complete muscular relaxation. Rarely (e.g., with peroneus) an open operation to suture the ruptured ligaments and tendon sheath is desirable.

DIVISION OF MUSCLES AND TENDONS.

1. CONTRACTION-RUPTURE. Voluntary, from an excessive or ill-balanced purposive movement, e.g., the rectus abdominis in labour. Involuntary, as in tetanus.
2. TRAUMATIC. Contusions, e.g., the extensor tendons of the fingers by a sharp blow over the knuckles. Wounds dividing muscles and tendons.

POSITION OF RUPTURE. (*a*) In the muscle belly; (*b*) At the junction of belly and tendon; (*c*) In the tendon; (*d*) At the insertion of the tendon where a piece of bone may be broken off.

MUSCLES COMMONLY AFFECTED.—Sternomastoid by traction during labour. Biceps cubiti (long head). Flexor tendons of the fingers and thumb by stab and incised wounds. Extensor tendons of the fingers by contusions. Muscles of the back and rectus abdominis by violent strains, tetanus, and parturition. Tendon of the adductor longus in riding. The quadriceps tendon or the ligamentum patellæ by contraction. The inner head of the gastrocnemius, the tendo achillis, or the plantaris in athletic exercises (tennis leg).

SIGNS.—Sudden sharp pain. Snapping sound. Loss of function. Swelling and ecchymosis, with a palpable gap if the muscle belly is affected.

RESULTS.—Recovery if treatment is good and the muscle belly affected. Permanent disability if the tendons are divided and retract. Stiffness from adhesions of the tendons to the sheath. Weakness from the stretching of a fibrous scar.

Traumatic Affections of Muscles, *continued.*

TREATMENT.—In slight cases, immobilization in a position of extreme relaxation of the affected muscle.

In most cases, union by suturing. If the gap is very long: (*a*) Turning back from each end a tendon flap; (*b*) Grafting the distal tendon into a neighbouring tendon with similar function; (*c*) Filling the gap by strands of catgut or piece of tendon taken from elsewhere.

Inflammation and Degeneration of Muscles.

1. SIMPLE MYOSITIS, resulting from a wound or contusion.
2. RHEUMATIC MYOSITIS, causing pain and stiffness, e.g., stiff neck or lumbago.
3. ACUTE SUPPURATIVE MYOSITIS arises by infection or in pyæmia. Results in considerable cicatricial deformity.
4. CHRONIC TUBERCULOUS MYOSITIS, secondary to bone disease, e.g., psoas abscess.
5. SYPHILITIC MYOSITIS from the formation of gummata, or fibroid thickening. Most often seen in the sternomastoid.
6. PARASITIC MYOSITIS. Trichinosis.

The adult trichina worm lives in the alimentary canal of pigs, rats, and other usually carnivorous animals. It measures 1·5 mm. (male) to 3 mm. (female) long. Pigs become infected by eating dead rats, by being fed on offal, and from one another. The embryo worms work their way from the alimentary canal through the peritoneum to the muscles. The diaphragm, rib, and trunk muscles are most affected. When it reaches the muscle, the embryo becomes encysted between the fibres in an oval cyst ·4 mm. long. In this condition their vitality is very resistant to heat, cold, or pickling.

CLINICALLY, the disease occurs in epidemics in Germany and America. Three stages are recognized: (*a*) A stage of enteritis with severe colic and diarrhœa, lasting about 10 days. (*b*) A febrile stage, temperature rising to 102–106° F., with severe muscular pains and swellings and some œdema; (*c*) A stage of subsidence, which occurs 6 weeks after infection. It is liable to be mistaken for enteric fever.

7. MYOSITIS OSSIFICANS.

(*a*) TRAUMATIC. A muscle after injury, in the process of repair becomes ossified. It then forms a hard, sharply-defined mass. This is seen most often in the adductor longus, and is known as the rider's bone. Also in the quadriceps after injuries of the knee. In the former case removal may be necessary. In the latter the X rays are useful in diagnosis, and nothing can be done by treatment.

(*b*) IDIOPATHIC. This affects many muscles, chiefly those of the back and trunk. It generally attacks young men, and is of slow and steady course, resulting in extensive ankylosis.

8. MYOSITIS FIBROSA, or the fibrous degeneration of muscles.

ISCHÆMIC PARALYSIS is the common name for this, which was first described as VOLKMANN'S CONTRACTURE.

CAUSES. Tight splints and bandages, Esmarch's bandage, arterial thrombosis.

POSITION. It has generally been observed in the forearm, and in the great majority of cases has occurred in the treatment of fractures.

PATHOLOGY. The muscles are deprived of their blood supply by pressure, and this may act further by producing thrombosis. The muscle fibres then suffer rigor mortis and gradually disappear, and are replaced by fibrous tissue, which contracts. A diminution in the blood supply to the growing ends of the bones probably also takes place, and accounts for the shortness of the affected arm.

SYMPTOMS. Great swelling of the limb, with perhaps sloughing from pressure, occur at the time, and within a few days the paralysis is noticed.

Flexion of the fingers at the phalangeal joints. These can only be extended when the wrist is flexed, showing that the flexor muscles are too short.

Pronation of the hand is permanent from contraction of the pronator radii teres.

Some flexion of the elbow is caused by contraction of the flexor muscles which arise from the humerus.

Wasting is very marked, and is due to the atrophy of the muscles.

The bones of the forearm are ½ in. to 1 in. shorter than on the other side in cases where the condition has arisen in growing children.

Anæsthesia may be present, but has an irregular distribution; the hand is cold, the skin blue and shiny, but trophic ulceration is seldom seen.

Electrical reactions are generally merely diminished, but rarely the reaction of degeneration occurs. In this case, as in those showing anæsthesia and trophic lesions, the nerves have probably been affected as well as the muscles.

DIAGNOSIS must be made from

PRIMARY NERVE LESIONS, especially musculospiral paralysis. Here there is no flexor but an extensor lesion. In all nerve lesions the paralysis is accompanied by anæsthesia, and both follow the anatomical distribution of the nerve. Reaction of degeneration is present.

INFANTILE PARALYSIS. There is no local lesion. Reaction of degeneration is well marked, and contraction is much slower in its onset.

SYNOVITIS AND INJURY. In this matting and contraction of the tendons may occur. The fingers cannot be extended when the wrist is flexed.

Ischæmic Paralysis, *continued.*

Prognosis.—Is bad in proportion to the extent and duratio without treatment of the affection. If the condition recognized early, the deformity can be prevented or remove by systematic exercises.

Treatment.—Massage with passive movements, continue daily for two years.

Operations.—Cutting and lengthening the flexor tendon a very difficult and tedious operation. Removal of piece from the middle of the radius and ulna. This best done by oblique incisions, and the bones then wire to prevent fibrous union. This shortening of the bon compensates the relative shortness of the flexor muscles.

Tumours of Muscles.

PRIMARY.—Sarcoma, fibroma (and gumma).

Sarcoma is round- or spindle-celled. At first it forms a roun encapsulated mass. But it grows rapidly and becom diffused beyond the capsule. Wide excision or amputatio is required. Any solid muscular swelling which grow steadily in spite of iodides should be regarded as sarcomatou

SECONDARY.—Carcinoma or sarcoma.

DISEASES OF THE TENDON SHEATHS.

Tenosynovitis.

1. ACUTE SIMPLE. From strains and sprains. Commonest i extensor muscles of the thumb and peroneal of foot.

   Signs : Pain, swelling, and fine crepitus when the tendon i moved.

   Treat as for a sprain.

2. ACUTE SEPTIC. Caused by septic wounds or extension fro neighbouring inflammatory foci. Constitutes the thecal variet of whitlow (p. 28). Is apt to cause sloughing of the tendo and permanent adhesions.

   Treat by incisions and passive congestion.

3. SIMPLE CHRONIC. Left after the acute attack, or it is cause by continuous over-strain. Increase of glairy effusion into th sheath, with swelling and weakness.

   Treat by counter-irritation and firm pressure. Rarely b puncture or incision.

4. CHRONIC TUBERCULOUS.—Common in the wrist and ankl Two conditions may occur, separately or together,—

   (*a*) Great swelling and hyperplasia of the synovial membran in which tuberculous granulations develop.

   (*b*) Effusion of glairy fluid and deposit of fibrin in the form " melon-seed " bodies.

   Signs are chronic doughy swelling, with weakness and slig pain. May spread to the bones and joints, or suppurate.

   Treat by rest and pressure. If this fails, open and scra out, rubbing in iodoform.

Ganglion.—A chronic synovial cyst, generally connected with a tendon sheath.

OCCURS generally at the back of wrist, connected with thumb or 1st finger extensor tendons. May occur on front of wrist or in the ankle region.

CAUSED BY a hernial protrusion of synovial membrane through the tendon sheath.

CONSISTS OF a firm, round, elastic, painless swelling over a tendon filled with fluid or colloid.

TREATMENT. (1) Subcutaneous rupture by pressure; (2) Puncture followed by pressure; (3) Excision.

COMPOUND PALMAR GANGLION is a tuberculous synovitis which affects the synovial membranes of the flexor tendons of the fingers and thumb under the anterior annular ligament. It forms a fluctuating swelling in the palm and above the wrist, which may extend up the thumb and little finger. It contains (1) Tuberculous granulations growing from the synovial membranes; (2) Some free fluid; (3) Fibrinous melon-seed bodies.

TREATMENT by rest, and firm pressure with Scott's dressing, and if this does not succeed, incision, scraping, and iodoform inunction. Permanent stiffness from tendon adhesions is likely to follow.

DISEASES OF BURSÆ.

Bursitis.

1. ACUTE SIMPLE BURSITIS.—From injury or irritation.

   TREAT by rest and fomentations, and aspiration if necessary.

2. ACUTE SEPTIC BURSITIS.—From septic wounds, or infection of a simple bursitis.

   TREAT by incision and drainage.

3. CHRONIC SEROUS BURSITIS.—The bursa is enlarged and distended by chronic effusion. Very common in the prepatellar bursa, forming the "housemaid's knee"; over the acromion, "the deal-runner's shoulder"; over the ischial tuberosity, the "weaver's bottom." It is caused by constant irritation and pressure. In course of time the walls become thick and a plastic or fibrous bursitis arises.

   TREATMENT.—(1) Pressure and counter-irritation—this is only likely to succeed temporarily and in thin-walled recent cases; (2) Pressure after aspiration—recurrence is the rule; (3) Aspiration and injection of iodine—uncertain and painful; (4) Excision—the only satisfactory treatment in really chronic cases.

4. CHRONIC PLASTIC BURSITIS.—Inside the sac becomes thickened by fibrous tissue and nodular masses, some of which may form free bodies.

5. FIBROID BURSITIS.—The sac becomes converted into a solid fibrous mass with small central cavity.

   THE TREATMENT for this and No. 4 is only excision.

Bursitis, *continued.*

6. TUBERCULOUS BURSITIS.—Similar to synovial tuberculous changes elsewhere. Usually leads to chronic suppuration.
   TREAT by excision or scraping.
7. SYPHILITIC BURSITIS.—Very rarely in the form of a symmetrical serous affection in the secondary stage. Common as a gummatous disease in the late stage, when it is apt to burst and cause the characteristic gyrate ulcers over the joint.
8. GOUTY BURSITIS.—Very common over the big toe, frequent over the olecranon. Urate of soda forms a hard tophus which may lead to inflammation and discharge.
9. MALIGNANT BURSITIS.—Rarely a bursa becomes the seat of a sarcomatous growth. This is manifested by the nodular character and rapid growth of the tumour.
   FREE EXCISION or AMPUTATION will be required.

Special Bursæ liable to disease.

SUPRA-ACROMIAL: "the deal-runner's shoulder."

SUB-DELTOID, liable to be confused with shoulder-joint effusion (*q.v.*).

SUBSCAPULAR, may communicate with the joint and be affected in tuberculous disease of the articulation.

SUPRA-OLECRANON: "miner's elbow."

GLUTEAL BURSÆ: (1) Between the osseous and fascial parts of the insertion of the gluteus maximus; (2) Between the great gluteal tendon and the great trochanter. Causes abduction of the hip.

SUPRA-ISCHIAL, "the weaver's bottom."

SUB-PSOAS, generally bilateral, between the psoas tendon and the brim of the pelvis. May communicate with the joint. Causes flexion of hip with pain on extension, other movements being free.

PATELLAR BURSÆ. These are four in number, though only one is commonly diseased.

- (1) Suprapatellar. The bursa most commonly affected with all varieties of disease constituting "housemaid's knee."
- (2) Subquadriceps. This usually communicates with the knee-joint and is affected with it.
- (3) Supraligamentous, over the patellar ligament.
- (4) Subligamentous, under the ligament, and may press into the joint itself.

SEMIMEMBRANOSUS.—Between the inner head of origin of the gastrocnemius and the insertion of the semimembranosus. It is tense on extension and lax on flexion. It often communicates with the joint, and forms the commonest variety of "Baker's cysts."

SUBSARTORIAL.—Between the insertion of the sartorius and the inner tibial tuberosity, associated with the insertions of the semitendinosus and gracilis.

SUB-ACHILLES.—Forms a swelling on either side of the tendo Achillis.

ADVENTITIOUS BURSÆ.—Form over any normal or abnormal bony prominence. The commonest are over the vertebra prominens of the neck, between the hyoid bone and thyroid cartilage, the inner femoral condyle in genu valgum, the inner or outer sides of the foot in talipes valgus or varus, the metatarsophalangeal joint of the great toe in hallux valgus. The last is known as a BUNION.

CHAPTER XVII.

DEFORMITIES.

Torticollis—Wry Neck.

ANATOMY. — Contraction of sternomastoid (constant); of trapezius, deep posterior cervical muscles, cervical fascia (often). Head inclines to affected side. Face turns to unaffected side. Atrophy of affected side of the face. Primary curve of cervical spine. Secondary curve of dorsal spine. Secondary shortening of ligaments and moulding of bones and joints.

VARIETIES.

- CONGENITAL. From an intra-uterine lesion.
- MUSCULAR. From laceration of muscles at birth or contraction after disease. Rheumatic from myositis.
- SPASTIC. Clonic spasms of affected muscles; due to reflex or central irritation of cortical centres; young adults, generally women, with epileptic history.
- PARALYTIC. From paralysis (infantile generally) of the opposite muscles.
- NEUROTIC. In hysterical women.

DIAGNOSIS from CARIES OF SPINE (tenderness and pain on movement). DEEP ABSCESS of the neck. RHEUMATIC INFLAMMATION. By absence of pain, and tense band formed by the sternomastoid.

TREATMENT.

- SLIGHT CASES: Massage, retentive apparatus.
- CASES WITH WELL-MARKED CONTRACTURE: Tenotomy of sternomastoid and fascia. Open incision across origin of sternomastoid. Free division of the muscle and any bands of fascia. Retention apparatus for a few weeks. Massage and exercises.
- SPASTIC CASES: First try general treatment.
  - Operation: Excision of parts of spinal accessory and posterior primary divisions of upper three cervical nerves.

Scoliosis.—Lateral curvature of the spine, with rotation of vertebræ.

CAUSES.

- CONGENITAL.—From presence of a half vertebra.
- RICKETS.—Early or late.
- STATIC CAUSES. (To compensate for another deformity)—Torticollis—Empyema—Hip disease or dislocation—Any shortening of the lower limb.
- ADOLESCENCE.—Anæmia and asthenia during rapid growth. Muscles and ligaments stretch. Especially in occupations with much standing, lifting, or carrying.

ANATOMY.

VERTICAL CURVES. Usually main curve with convexity to the right occupying the dorsal region. Secondary curve to the left in lumbar region.

ROTATION. Due to greater displacement of bodies than of the laminæ of the vertebræ.

Bodies will be directed to the convexity, spines to the concavity of the curve.

The ribs will project on the right side posteriorly, and be flattened on the opposite.

The ribs will project on the left side anteriorly.

The sternum remains in the mid line, but is twisted to the right.

Right shoulder projects behind and is raised (" shoulder grows out ").

Left hip appears more prominent.

SECONDARY CHANGES.—Vertebræ and discs become shorter, and the ligaments and muscles are shortened on the side of the concavity. Joints become ankylosed.

STAGES OF THE DEFORMITY.

1. The deformity can be rectified by the patient's own muscular effort.
2. Can be rectified when the spine is flexed, or by manipulation, e.g., suspension.
3. Deformity becomes fixed by the shortening of muscles and and ligaments and by ankylosis.

DIAGNOSIS.—Primary deformities or diseases of shoulder, breast, or hip must be distinguished from similar deformities secondary to scoliosis.

PROGNOSIS.—Depends on the stage the deformity has reached. When it can be reduced by manipulation the prognosis is good. When it cannot be altered it cannot be cured, but may be arrested.

TREATMENT.—Remove the cause in static cases. Treat general conditions, e.g., rickets or anæmia.

In adolescent cases.

Rest on an inclined plain.

Massage and cold douches.

Exercises : Flexion and extension of the back when standing. Horizontal bar exercises. Flexion and extension of the back when lying face down at the end of a table.

Carefully applied Chance's spinal support.

Kyphosis.—Abnormal backward curve of the spine. Usually limited to the dorsal region, unless it follows organic spinal disease. There are three groups of cases :—

1. DEFECTIVE GROWTH OR HABIT : Rickets in children—Adolescence—Occupations which necessitate stooping—Senile atrophy of muscles of the back.
2. From GENERAL DISEASE of the spine and its muscles, etc. : Osteoarthritis — Osteitis deformans — Osteomalacia—Acromegaly—Pulmonary Osteo-arthropathy.
3. From LOCAL SPINAL DISEASE : Fractures—Pott's disease—New growths.

Kyphosis, *continued.*

Treatment. (In the first group only).

Remove the cause, if possible (e.g., myopia or any stoopin habit).

Massage, electricity, and exercises for the back.

Recumbency on a hard, flat mattress or board.

Chance's splint.

Lordorsis.—An abnormal forward curve of the spine, usuall in the lumbar region.

It is always secondary to: (1) Diseases of the hip or of th psoas muscle. A primary kyphotic curve above. (2) An abnormal growth which places the centre of gravity forward e.g., pregnancy.

Deformities of the Upper Extremities.

SPRENGEL'S SHOULDER.—Congenital deformity. The vertebr border of the scapula is raised, the serratus and trapezius muscle are ill-developed.

CLUB-HAND.—Usually associated with congenital absence of th radius.

POLYDACTYLISM.—Supernumerary fingers or toes: usually onl affects the phalanges.

ECTRODACTYLISM.—Absence of one or more digits.

MACRODACTYLISM.—Overgrowth of one or more digits.

SYNDACTYLISM.—Fusion of adjacent digits, either complete o only at their bases—the so-called webbed fingers or toes. Th same condition may be acquired after burns.

Treat by a plastic operation.

CONGENITAL CONTRACTION OF THE FINGERS.—Usually o the little finger only. The last two phalanges are flexed, th first extended. The central digital prolongation of palmar fasci is contracted.

Treat by the division of this band.

SPRING OR SNAP-FINGER.—Development of a sesamoid bon in the flexor tendon, which catches under the tendon sheat opposite the metacarpo-phalangeal joint.

MALLET- OR HAMMER-FINGER.—Flexion of the termina phalanx. Acquired deformity from rupture of the extenso aponeurosis.

Treat by a palmar splint or suture of the tendon.

DUPUYTREN'S CONTRACTION.—An acquired flexion of th fingers.

Usually in Gouty men; often Symmetrical, often Hereditar

Constant Pressure on the palm by an instrument ma cause it.

Affects the little and ring fingers first, the others later.

The flexion is of the first and second phalanges, the third i extended.

An indurated nodule forms over the palmar fascia.

Consists of a contraction of the palmar fascia and its lateral prolongations into the digits.

DIAGNOSIS from congenital contraction (above) and contraction of the flexor tendons, which is released on flexing the wrist.

TREATMENT by incision or excision of the contracted bands.

Coxa Vara.—Deformity of the hip joint due to a lessening of the angle between the neck and shaft of the femur.

The angle is normally about 125°; at birth it is much more open (160°), and continues to diminish until growth is complete at 18–20. In late life atrophy of the neck may lead to further closing of the angle.

CAUSES AND VARIETIES.

RICKETS.—In young children. Bilateral. Very rare.

ADOLESCENCE.—The common form. The normal closure of the angle at the neck of the femur is carried too far.

Carrying heavy weights and late rickets may also be concerned in this form.

SLIPPING OF THE EPIPHYSIS.—Accounts for cases coming on after an injury. The deformity is marked and unilateral.

SENILE FORM.—Atrophy of the neck makes the bone yield. Usually associated with osteo-arthritis.

ANATOMY.—The angle between the neck and shaft is reduced to a right or even an acute angle. The neck of the bone is shorter than normal. The margins of the head are overgrown. The back of the femoral neck is more absorbed than the front, so that the shaft is everted.

SYMPTOMS.

Aching and tiredness, with marked limp. Shortening of the leg, the trochanter rising above its normal position and becoming very prominent. Eversion of the leg at the hip joint. Adduction very marked, especially on flexing the thigh. Scissor-leg deformity if both sides are affected. Abduction and internal rotation are very limited. Other movements of the hip are free and painless. There is a marked absence of tenderness on pressure.

DIAGNOSIS from tuberculous disease of the hip—Congenital dislocation—Mal-united fracture (*see* " Hip Disease ").

TREATMENT.

REST IN THE EARLY STAGES when the deformity is still in progress.

SUBTROCHANTERIC OSTEOTOMY, after growth has ceased. The limb should be put up in an abducted position. Later a tilting of the pelvis will make up for much of the shortening.

Coxa Valga.*—A very rare condition. The opposite to vara, i.e., the femoral angle is more widely opened, and varies from 130° upwards to 180°.

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* Tubby, *Proceedings of the Royal Society of Medicine*, March, 1908.

Coxa Valga, *continued.*

CAUSES.

CONGENITAL.—Frequently found in congenital hip disease. It may be the cause or result of the dislocation.

ACQUIRED.—Any hanging limb (e.g., paralysis) or stump will acquire a straight-necked femur. Infantile paralysis, scoliosis, or other static causes. Mal-union after fractures. Rickets (very rare).

May be unilateral or bilateral. In the latter case it is probably a slight degree of congenital dislocation.

SYMPTOMS AND SIGNS.

Limping towards the affected side: the gait is rolling in bilateral cases. Lengthening of the limb to 2–3 cms.

Abduction and external rotation of the leg, whilst adduction is limited. Region of the trochanter is flat, and the trochanter is below Nélaton's line. Skiagram shows the straight neck.

TREATMENT, by subtrochanteric or cervical osteotomy.

Genu Valgum—Knock-knee.

CAUSES.

RICKETS, the common cause in young children.

STATIC CAUSES, i.e., long standing in a rapidly-growing patient whose ligaments are soft and relaxed.

TRAUMATIC CONDITIONS which interfere with the position or growth of the condyles or tibial tuberosities.

ANATOMY.—One or more of three changes have occurred :—

1. The lower end of the femur is bent outwards.
2. The upper end of the tibia is bent inwards.

   These two conditions are most common in rickets.
3. The internal condyle of the femur and internal tuberosity of the tibia are developed disproportionately to the external parts. The common condition in adolescents.

   The internal lateral ligament is relaxed, and in many cases the internal tibial tuberosity can be separated from the femur by forcible abduction.

   The external lateral ligament, iliotibial band, and biceps tendon are proportionately shortened.

   The patella is displaced towards the outer side of the knee.

PATHOLOGY.—In standing, especially with the feet apart, the line of the tibia lies outside that of the femur, the internal lateral ligament of the knee is relaxed, the external half of the joint is pressed together, the internal half is pulled apart. Hence the internal ligament stretches and the internal parts of the joint grow faster than the external.

SIGNS.—When the knees are extended and touching each other the ankles are widely separated.

With very slight flexion of the knees the ankles come together in the majority of cases. This is due to the fact that flexion is always accompanied by some internal rotation which swings the feet round towards one another.

The toes point outwards when standing. This is due to an exaggerated external rotation.

When walking the knees are kept slightly flexed, so as to minimize the deformity, which, as above explained, tends to disappear on flexion.

A bursa often forms over the inner sides of the knees when they knock together.

It is usually bilateral. Rarely it may be unilateral, or one knee may be in genu valgum and the other genu varum.

COMPLICATIONS.—Scoliosis or flat-feet often co-exist in genu valgum. The pelvis is tilted in unilateral cases.

TREATMENT.

1. REST WITH SPLINTS.—In all rickety cases—In all patients below the age of puberty—In unhealthy patients when the condition is progressing.

   Young children should be kept off the ground, either in bed or by the application of splints which extend beyond the feet.

   Long outside splints should be re-applied daily, with massage night and morning.

   Starch bandages should only be used to fix the splints for one or two weeks at a time at first, to get the child used to them, or in cases where no one can be trusted to re-apply them.

   Iron splints fixed into the boots, with straps to fasten above and below the knee for adolescent cases.

2. OPERATION.—For all cases in which growth has ceased and in which the deformity is permanent.

   (*a*) OSTEOTOMY OF THE SHAFT OF THE FEMUR (Macewen's operation).—The bone is partly cut and partly broken just above the condyles. The limb is put up in a rectified position.

   This is the easiest and best operation in most cases.

   (*b*) OSTEOTOMY OF THE INTERNAL CONDYLE (Ogston's operation).—The internal condyle is separated from the external by an oblique incision into the joint. The loose condyle with the tibia is then slid upward.

   This is seldom performed, because of the risk of causing arthritis with adhesions.

   (*c*) CUNEIFORM OSTEOTOMY OF THE TIBIA.—A wedge-shaped piece is cut out from the shaft of the tibia on its inner side below the tubercle.

   This is probably the most accurate method, but it requires some precision.

Genu Varum—Bow Legs.—Is similar, but opposite to the above. All the changes on the inner side of the joint above described are here external, and *vice versa*. It is much rarer than genu valgum, It seldom requires treatment, as it causes less inconvenience.

When TREATMENT is required it is on the same lines as above, femoral osteotomy being the operation of choice.

TALIPES (CLUB-FOOT).

Causes.

CONGENITAL.—Often hereditary. Malposition in utero. Recurrence to a simian type.

ACQUIRED.

NERVE DISEASE: Anterior poliomyelitis. Cerebral and spinal sclerosis, producing spastic paralysis. Lesions of peripheral nerves.

MUSCULAR DISEASE: Suppuration and contraction in muscles and fasciæ.

BONE INJURIES: Fracture and dislocations with bad union about the ankle.

COMPENSATORY to other deformity.

STATIC CAUSES: Prolonged standing—Decubitus.

Varieties.

TALIPES EQUINUS.—Hyperextension of the ankle. Walks on the toes.

TALIPES CALCANEUS.—Flexion of the ankle. Walks on the heel.

TALIPES VARUS.—Adduction of the foot at midtarsal joint. Inner margin of the sole raised. Walks on outer side of foot.

TALIPES VALGUS.—Abduction of the foot at midtarsal joint. Outer margin of the sole raised. Walks on inner side of the foot.

EQUINO VARUS, CALCANEO VALGUS—the commonest combinations of the above.

EQUINO VALGUS, CALCANEO VARUS—rare.

General Features of Congenital Talipes.—Exists from birth. Often bilateral. Is associated with primary malformation of bones and ligaments and contraction of muscles. Except in early infancy the deformity cannot be corrected by simple manipulation. No wasting or spasm of muscles. No trophic changes or defects in the circulation. Electrical reactions of the muscles are unaltered. Growth of the limb is not impaired. Furrows are formed in the flexures of the foot.

Treatment of Congenital Talipes.

IN INFANCY.—Daily manipulation, massage, and bandaging.

AFTER THE CHILD HAS BEGUN TO WALK.—Bandaging to a malleable splint in the deformed position, and then bending towards the correct attitude.

IN CHILDHOOD when the deformity cannot be improved by manipulation.—Tenotomy of contracted muscles. Fixing in corrected position by plaster. Massage and passive movements.

IN ADULT CASES.—Some form of osteotomy.

General Features of Paralytic Talipes.—Begins in second or third year with infantile paralysis. Seldom bilateral. Limb is cold, blue, and clammy. Muscles are much wasted and paralyzed. Electrical reaction absent in paralyzed muscles. General growth of the bones diminished. The limb is flabby, and can easily be restored to the correct position until late contractions have developed. Furrows do not form in the flexures.

Treatment of Paralytic Talipes.

IN EARLY STAGES.—Massage and electrical treatment of the paralyzed muscles. Retention of the foot in position which relaxes the paralyzed muscles.

IN CASES WHERE NO FURTHER MUSCULAR RECOVERY IS POSSIBLE.

- WHERE DEFORMITY HAS NOT BEEN PREVENTED:
  - Tenotomy of muscles opposed to the paralyzed groups.
  - Grafting parts of unparalyzed tendons into those which are paralyzed.
- WHERE ALL THE MUSCLES ARE PARALYZED, and in many of the above cases as a sequel to the other methods:
  - A BOOT with irons and springs.
  - Or an EXCISION, with fixation, of a flail joint or one in bad position.

General Features of Spastic Talipes.—The symptoms of spinal sclerosis or cerebral disease are present. The deformity can be reduced by manipulation, except in very old cases. Exaggerated knee-jerks and ankle clonus. The muscles do not atrophy until late.

TREATMENT.—Massage and galvanism. Some retentive apparatus.

Talipes Equinus.

CAUSES.—Nearly always acquired. Infantile paralysis of anterior tibial muscles. Partial paralysis of all leg muscles, the calf muscles overcoming the anterior. Spastic contraction of the calf muscles. Compensatory to any shortening of the leg. From pressure of the bedclothes.

SIGNS. Three degrees of extension of the ankle:—

1. Foot cannot be flexed beyond a right angle. Thus the toes catch the ground in walking.
2. The heel cannot be brought to the ground. Patient walks on the balls of the toes.
3. Foot is in a line with leg or a little behind it. Toes are doubled under the foot, and patient walks on their dorsal surfaces.

   Pes cavus is often present from a flexion of the sole at the mid-tarsal joint, especially in paralytic cases.

TREATMENT.—Tenotomy of the tendo Achillis. Plaster-of-Paris in over-corrected position.

IN PARALYTIC CASES.—Grafting half tendo Achillis into the tibialis anticus, and the peroneus longus into extensor tendon. A boot with a toe-raising spring.

COMPENSATORY TALIPES EQUINUS should not be treated unless the primary condition is cured.

Talipes Equino-Varus.

CAUSES.

CONGENITAL.—Represents a reversion to a simian type. In infants the head and neck of the astragalus form an angle of 35° inwards, with the body. If this persists or is exaggerated, adduction of the foot with varus results.

ACQUIRED.—Paralysis of extensor and peroneal muscles. Tibialis anticus may escape or not. Over-action of calf muscles and tibialis posticus, and sometimes of tibialis anticus. Injury of the external popliteal nerve, *p.* 108.

SIGNS.—Heel cannot be brought down to the ground. Foot is adducted at the mid-tarsal joint. Patient walks on the outer margin of the sole. A transverse furrow crosses the sole at the mid-tarsal joint, and a longitudinal furrow runs along the sole in congenital cases. A callosity or bursa is formed over the cuboid. A secondary contraction occurs of plantar fascia.

ANATOMY.

ASTRAGALUS is misshaped. The head is set at an angle of 50° with the body. It is displaced forwards and inwards from the ankle joint.

SCAPHOID is on the inner side of the astragalus and almost touches the internal malleolus.

THE LIGAMENTS on the sole and inner side of ankle are concontracted, viz.: Internal lateral ligament, inferior calcaneoscaphoid ligament, plantar fascia.

THE FOLLOWING MUSCLES ARE CONTRACTED: Tibialis posticus, tibialis anticus, flexor longus hallucis, flexor longus digitorum, abductor hallucis, tendo Achillis.

TREATMENT.

IN SLIGHT OR EARLY CASES.—Massage, manipulation, and bandages. Malleable splints.

IN CASES WHERE CONTRACTION OF THE MUSCLES AND LIGAMENTS HAVE OCCURRED.

1. Tenotomy of tibialis posticus and anticus. Division of internal lateral ligament in its anterior part. Division of the plantar fascia.

   All these may be divided by a single open incision which also cuts the spring ligament. This is Phelps' operation.

2. Subsequent tenotomy of tendo Achillis.

IN CASES OF OLD STANDING, WHERE ANKYLOSIS AND BONY CHANGE IS MARKED, or where other measures have failed. —Excision of astragalus, or wedge-shaped tarsectomy from the outer side of foot.

IN PARALYTIC CASES.—After correcting the deformity by tenotomies, graft half the tendo Achillis into the peroneus longus, and the tibialis anticus into the peroneus brevis.

Talipes Calcaneus.—May be either congenital or paralytic. Sometimes follows a too free tenotomy of the tendo Achillis. The patient walks on his heel, the gait is heavy and stamping. The extensor tendons are contracted. The tendo Achillis is stretched or the posterior muscles are paralyzed.

TREATMENT.

In Congenital Cases.—Tenotomy of the extensor tendons.

In Paralytic Cases.—Grafting peroneus longus tendon into tendo Achillis. Boot with a toe-depressing spring.

In Traumatic Cases.—Shortening of the tendo Achillis, or transplanting the tuberosity of the os calcis lower down.

Talipes Valgus.—Rare except in conjunction with flat-foot. May be congenital or paralytic. There is abduction of the foot at the mid-tarsal joint and an eversion of the sole of the foot. The peronei muscles are contracted in congenital cases. The tibials and some of the extensors are paralyzed in paralytic cases.

Treatment. Division of the peronei tendons in congenital cases. Grafting of part of the tendo Achillis into the tibialis posticus in paralytic.

Flat-Foot—Pes Planus—in a simple form consists in a mere loss or flattening of the arches of the foot, but it is nearly always combined with some degree of valgus, i.e., of abduction and eversion of the front part of the foot.

CAUSES.

Static Conditions, i.e., long standing in growing persons.

Congenital Conditions: very long feet.

Paralytic.—Paralysis of the tibials and extensor muscles.

Inflammatory.—Rheumatic or gonorrhœal affections of the foot rapidly soften the ligaments and the arches of the foot give way.

Traumatic. —After Pott's fracture, or severe sprains or injuries of the ankle, there is a great tendency for flat-foot to be left.

ANATOMY.

1. The Longitudinal Arch of the foot is flattened. The muscles, ligaments, and tendons in the sole are stretched, viz.: The inferior calcaneoscaphoid or spring ligament. The calcaneocuboid ligaments (plantar ligaments). The tendons of the tibialis posticus and anticus. The flexor longus digitorum and hallucis with short muscles.

   The giving way of the calcaneoscaphoid ligament allows the astragalus to drop, so that it may actually touch the ground.
2. The Transverse Arch is flattened, the foot being broader than normal.
3. Abduction of the Foot, chiefly at the mid-tarsal joint. The astragaloscaphoid joint projects prominently on the inner side of the sole.
4. Eversion of the Sole of the Foot.—This also takes place at the mid-tarsal joint. The inner margin of the sole is depressed, the outer raised. The peronei tendons are tense and rigid.

Flat-foot, Anatomy, *continued.*

5. EVENTUALLY in old, neglected, and inflammatory cases the bones become distorted by osteophytic outgrowths, and the joints, especially the astragaloscaphoid, obliterated by ankylosis.

SYMPTOMS.—Walk is shuffling or waddling. The toes are turned outwards (splay-feet). The heels do not leave the ground before the toes. The whole sole comes into contact with the floor. All power of jumping or dancing or raising the body on the toes is lost or diminished. Aching pain on the sole of the foot over the mid-tarsal joint, much worse on standing or walking.

DEGREES OF MUSCULAR AND ARTICULAR CHANGE.

1. A MERE LOSS OF STRENGTH AND TONE IN THE MUSCLES, the ligaments being stretched. The patients can rectify the deformity to a great extent when they place their toes together and stand on tiptoe. The deformity can be easily rectified by passive movements.
2. ACTUAL PARALYSIS OR FUNCTIONAL INCAPACITY of the muscles, with contraction of the peronei. The patient cannot stand on his toes, or in any way restore the arch of his foot. Passive movements will, however, rectify this deformity.
3. ADHESIONS AND CONTRACTIONS fix the deformity so that neither active nor passive movements have any effect on it.
4. ACTUAL BONY DEFORMITY AND ANKYLOSIS render the condition permanent.

TREATMENT.

1. FOR THE FIRST DEGREE.—Massage with tip-toe exercises. Boots with stiffening on the inner side of the uppers and thickened inner border to the sole. A valgus pad or steel brace to support the instep, worn inside the boot.
2. FOR THE SECOND DEGREE, in addition to the above.—The peronei tendons have sometimes to be cut. The foot should be over-corrected and fixed for one month at a time in plaster bandages. It is useless to attempt the exercises until some tone has been restored to the muscles. A boot with an outside leg-iron and T-strap.
3. FOR THE THIRD DEGREE, especially in inflammatory cases.—Tenotomy of the peronei (where necessary). Wrenching the foot into good position under an anæsthetic. Fixing in a corrected position in plaster-of-Paris. A boot with a leg-iron must be worn for some time.
4. FOR THE FOURTH DEGREE.—Some form of tarsectomy. Removal of a wedge-shaped piece from the inner border of the sole. Removal of parts of the astragalus or scaphoid, or both.

Pes Cavus or Claw-foot.—The longitudinal arch of the foot is exaggerated. There is some degree of hammer-toes. It usually accompanies a slight degree of talipes equinus. There is more or less pain and disability on walking.

TREATMENT.—Tenotomy of the tendo Achillis (in equinus cases). Fasciotomy of the plantar fascia.

Hallux Valgus.—Outward displacement of the great toe. Is often caused by pointed-toed boots. The metatarso-phalangeal joint becomes very prominent, and is usually affected with hypertrophic osteo-arthritis. A marked bunion, i.e., a chronically inflamed bursa, forms over the prominent joint. The toes are crowded together, hammer-toe often co-existing in the other toes.

TREATMENT.—A straight inner-edged boot with a toe-post from the sole to keep the toe in position, for early cases.

A spring along the inner side of the foot drawing the great toe inwards by a band.

Excision of the joint and the bunion is the best treatment of most cases.

The external metatarsal condyle may be separated, and then slid back and pegged, after cutting the internal lateral ligament.

Hallux Rigidus or Flexus.—A flexion of the first phalanx on the metatarsal of the great toe. The joint is affected by osteo-arthritis. It often accompanies flat-foot.

TREATMENT is first that of the flat-foot, and then if necessary an excision of the head of the metatarsal bone.

Hammer-toe.—Hyperextension of the first phalanx, with flexion of the other phalanges. The first interphalangeal joint forms an upward projection. The point of the toe is on the ground. Corns or bursæ are formed under the head of the metatarsal bone and the point of the toe, and over the interphalangeal joint. The second toe is most frequently affected. The extensor tendons stand out very prominently.

CAUSES.—Sometimes congenital. Secondary to talipes equinus and pes cavus, or hallux valgus. Paralysis of the interossei and lumbricales (?) Short boots with high heels and pointed toes.

ANATOMY. Partial dislocation of the second phalanx downwards. Marked shortening of the lateral digital bands of plantar fascia and of the lateral ligaments of this joint.

TREATMENT.—Excision of the head of the first phalanx.

A plantar splint to which the toe is bound down may be used in slight cases or after operation.

Metatarsalgia, or Morton's Disease.—Pain at the heads of the metatarsal bones. Probably a variety of flat-foot affecting chiefly the transverse arch. Possibly a direct pressure on the digital nerves results. It occurs especially in gouty or rheumatic subjects. The foot is broader than normal, and corns form on the sole beneath the heads of the metatarsal bones.

TREATMENT.—Rest and massage. Support by some kind of a valgus pad or brace.

Chapter XVIII.

FRACTURES.

Predisposing Causes.

INCREASING LIABILITY WITH AGE.

Activity of occupation.

Male sex. Except lower end of radius and head of femur.

MORBID BONE CONDITIONS.

GENERAL.—Atrophy: Senile, disuse, paralysis, tabes—Fragilitas—Rickets—Osteomalacia.

LOCAL BONE DISEASES. — Sarcoma — Carcinoma — Gumma—Caries or necrosis: Septic, tuberculous, syphilitic.

Exciting Causes.

DIRECT VIOLENCE.—Transverse in direction. Little displacement. Often compound or comminuted.

INDIRECT VIOLENCE.—Oblique or spiral in direction. Great displacement.

MUSCULAR ACTION.—Patella and olecranon, common. Other bones, rare.

Spontaneous Fractures.—Cases where the exciting cause is trivial and fracture determined by a pre-existing morbid bone condition. These are:

1. FRAGILITAS OSSIUM.—The commonest cause in children. An imperfection in the ossification, the only symptom of which is the liability to fractures. It manifests itself in children, who lose this tendency in adult life. It may occur in new-born children, where the fractures are usually multiple. Union is rapid and normal.
2. NERVE CONDITIONS. — Tabes, syringomyelia, general paralysis. Tabes is the commonest cause in adults. There is no demonstrable change in the bone; possibly the ataxia may be concerned in it. The fracture is remarkably painless. It unites rapidly if properly immobilized, but there is an exuberant callus formation.
3. ATROPHY.—The common cause in old people. May be senile or due to disuse or paralysis. The hard bone is replaced by fat-containing cancellous tissue.
4. OSTEOMALACIA.—Usually attacks women, and is then associated with pregnancy. The tendency to fractures is associated with a softening and bending of the bones.

5. FŒTAL AND SCURVY RICKETS.—Usually occurs at the epiphyseal junction in scurvy, and in the shafts of the long bones in fœtal rickets.
6. INFLAMMATORY BONE DISEASES, whether they produce caries or necrosis. Tubercle (commonly), septic and syphilitic disease (both rarely).
7. TUMOURS OF BONE.
    - (*a*) Sarcoma, usually primary, may be medullary or periosteal. The fracture will not re-unite.
    - (*b*) Carcinoma — always secondary. The fracture usually re-unites if it is immobilized.
    - (*c*) Gumma.
    - (*d*) Hydatid cysts.

Varieties of Fracture.

SIMPLE.—No communication with a wound.

COMPOUND.—Fracture communicates with a wound.

INCOMPLETE FRACTURE.—Bone not broken through.

Greenstick. Occurs in children, especially with rickets. Concavity of long bone is bent. Convexity is splintered.

Fissures of long bones, especially near joints.

Crushing of outer table of bones: Occurs in skull—Small cancellous bones.

COMPLETE FRACTURES may be :—

Transverse, especially when caused by direct violence.

Oblique, especially when caused by indirect violence.

Spiral, especially when caused by rotation.

Comminuted, when bone is broken into more than two pieces.

Impacted, when one fragment is driven into another.

Complicated, when combined with an injury of another important structure, e.g., artery or joint.

SEPARATION OF EPIPHYSES.—Common in patients under twenty. May occur up to twenty-five.

Causes.—Joint injuries which produce dislocation in older patients. Syphilis in Infants. Tubercle. Infective osteomyelitis.

Anatomy. Fracture runs through diaphyseal side of the ossifying cartilage. Fractured surface of the epiphysis is concave. The older the patient, the more likely is it to involve the shaft as well as the epiphyseal line. Periosteum closely attached to epiphysis is stripped off shaft. Shaft may be thrust through the periosteal sleeve.

Results: (1) Recovery as in fracture if apposition is good; (2) Retarded length of a long bone—deformity of wrist or ankle resulting from this; (3) Suppuration, with death of epiphysis; or (4) Arthritis from extension to joint.

Epiphyses Affected, in order of frequency:

Upper and lower ends of humerus, lower end of radius, lower end of femur, about equal.

Upper end of femur. Upper end of tibia, tibial tubercle alone, great trochanter, rare.

Signs of Fracture.

1. WOUND, CONTUSION, OR BRUISING
2. UNNATURAL MOBILITY in the shaft of long bone.
3. CREPITUS on movement.
   - May be SIMULATED by: Osteo-arthritis — Tenosynovitis — Surgical emphysema.
   - May be ABSENT in: Fracture with Impaction — Separated epiphysis—Wide separation of fragments—Interposition of soft parts—Partial fracture (greenstick).
4. DEFORMITY or displacement.
   - CAUSED BY: (*a*) Violence; (*b*) Weight of limb; (*c*) Muscular action; (*d*) Manipulation.
   - VARIETIES. — Angular — Longitudinal Divarication—Lateral—Rotatory—Depression.
   - EVIDENCES OF DISPLACEMENT.—Altered line of the bone—Altered measurement (generally shortened)—Altered relation of neighbouring bony points.
5. PAIN and loss of function.
6. SKIAGRAM showing fissure or fracture.

Symptoms following Fractures.

Shock, slight in uncomplicated cases.

Febrile reaction. Temperature 100° to 101° F. on second and third days. In proportion to ecchymosis.

Rare Symptoms.

Delirium tremens in drinkers.

Fat embolism: Dyspnœa — Syncope — Coma (from blocking of capillaries of lungs and brain with fat globules).

Complications of a Fracture.

1. COMMINUTION: leading to non-union, necrosis, or deformity.
2. JOINT INVOLVEMENT: Arthritis and adhesions.
3. DISLOCATION.
4. INJURY TO MAIN ARTERY: Aneurysm or gangrene.
5. INJURIES OF VEINS: Ecchymosis and œdema.
6. INJURIES OF NERVES. Rupture or involvement in callus.
7. INJURY TO VISCUS.

Repair of Fractures.

FIRST WEEK.

BLOOD-CLOT forms round the broken ends and beneath the torn periosteum. Soft parts become swollen and œdematous.

SECOND WEEK.

GRANULATION TISSUE replaces the clot. This is complete about the tenth day, when the fracture is fixed in a mass of granulation.

CALCIFICATION of the granulation tissue to form callus. Medullary tissue is also absorbed and replaced by callus. The ends of the bone become porous.

CALLUS is situated :—

1. Between periosteum and bone = external callus.
2. Between bony fragments = permanent or intermediate callus.
3. As a medullary plug = internal callus.

Calcification occurs first in the external, then in the internal, and last in the intermediate callus.

Amount of callus is in proportion to movement and irritation.

THIRD WEEK TO EIGHTH WEEK.

CONVERSION OF CALLUS INTO BONE.—Callus becomes more dense by further calcification which surrounds its vessels. Original bone becomes rarefied, and its vessels become continuous with those of the callus.

FINALLY. External and internal callus disappear by rarefying osteitis. Intermediate callus becomes condensed into hard bone by sclerosing osteitis, and this change spreads into the ends of the bone.

EXCEPTIONAL CASES.

IN IRREGULAR FRACTURES with much displacement. The angles between fragments are filled up—Projecting corners of bone removed—Architecture of the bone is remodelled.

COMMINUTED FRACTURES.—All the fragments are "set" in callus—Callus becomes densified and old bone rarefied until whole is welded into a homogeneous mass.

WHEN MOBILITY IS NOT PREVENTED.—Excessive external callus is formed — Sometimes cartilage occurs instead of bone.

FLAT BONES OF SKULL.—With no mobility little repair occurs—No callus is formed—Inner table is united most firmly.

ARTICULAR CARTILAGE is replaced by fibrous tissue ; rarely by cartilage.

RIB CARTILAGES are repaired by bony callus.

Microscopical Changes in the Repair of Fractures.—These are exactly similar to the repair of soft parts, with the addition of the processes of calcification and ossification. Thus :—

BLOOD-CLOT between the fractured ends.

HYPERPLASIA of tissues with cellular and plastic exudation.

Invasion of clot and replacement by LEUCOCYTES AND FIBROBLASTS.

VASCULARIZATION of this cellular layer by new vessels.

DEPOSIT OF LIME SALTS between the vessels.

Vessels thus lie in tubes of calcareous matter.

CALCAREOUS TUBES are lined by endothelial cells and covered by layer of osteoblasts, each derived from fibroblasts.

OSTEOBLASTS lay down lamellæ of true bone arranged concentrically round the vessels, some cells remaining between the lamellæ as Haversian cells.

Microscopical Changes in the Repair of Fractures, *continued.*

Large Fibroblasts called Osteoclasts erode the surfaces of the original bone and make it porous, and enlarge the Haversian canals by the same process.

Thus building up of new bone by osteoblasts in callus, and absorption of old bone by osteoclasts, proceed simultaneously and produce homogeneity.

Temporary callus is absorbed by osteoclasts removing the calcified granulation tissue.

Permanent callus is converted into true bone by osteoblasts.

Condensation of this and of the ends of the bone occurs by continuation of the same process.

Treatment of Simple Fractures.—Four cardinal rules :—

1. PREVENT FURTHER INJURY by "first aid."
2. SET THE BROKEN BONES in natural position.
3. FIX THE LIMB by retentive apparatus.
4. KEEP THE PART AT REST until union is firm.

REDUCTION OF THE FRACTURE.

Extension of the limb.

Manipulation of the broken ends.

Continuous Traction is necessary sometimes to overcome muscular spasm.

An Anæsthetic to abolish muscular spasm in all difficult cases.

Completeness of Reduction should be estimated by comparative measurement of sound side, or by a skiagram.

Time Limit for Reduction. Within the first week errors of reduction can easily be corrected. After the tenth day parts are "set" in calcified granulation tissue, and should not be moved.

FIXATION OF FRACTURES.

1. Splints.—Wood, metal, or leather.

   Ought to immobilize the joints above and below—Padded so as not to press too much on bony points—Wide enough to cover the limb—Applied in the position in which the part is to remain.

   Parts below the fracture should be bandaged to prevent swelling from œdema—Not so tight as to interfere with circulation.

2. Fixed Apparatus.—Starch bandages—Silicate bandages—Plaster-of-Paris : bandages or Croft's apparatus (flannel moulds of limb made in two parts and impregnated with plaster)—Leather splints first moulded on to a plaster cast of the limb (Hessing's splint).

   In each of these methods the part is first covered by wool or flannel bandages. The apparatus may be strengthened by metal strips.

3. Rest in Bed, with Extension sometimes: Fractures of Spine—Pelvis—Head and neck of the femur.

   Rest in bed with splints : Fractures of the thigh and leg.

4. Operation, with mechanical fixation of the fragments: (*a*) When reduction cannot be effected. (*b*) When reduction cannot be maintained—oblique fractures. Especially in bones of the leg. (*c*) In many compound fractures. (*d*) Fractures running into joints. (*e*) Complicated by injury of the artery or nerves. (*f*) Combined with a dislocation when the latter cannot be reduced otherwise. (*g*) In patella and olecranon.

Advantages of Operation: Accuracy of reduction. Complications can be dealt with. Excessive effusion can be removed. Massage and movement can be performed much earlier. Functional result is better.

Disadvantages.—Sepsis may occur and produce necrosis. Screws or wire may cause pain, irritation, or suppuration, and have to be removed later.

Methods.—Screws through oblique fractures. Silver wire, especially in patella and olecranon. Staples shaped like ⊓. Aluminium plates nailed on to surface of the bone.

After-treatment of Fractures.

MASSAGE.—Removes effusion. Prevents contraction of soft parts, atrophy of muscles, and adhesions in joints.

Without Removal of Splints:

Begin on second day in all possible cases.

With Removal and Re-application of Splints:

Begin from third to seventh day in impacted fractures near joints; in fractures of the neck of humerus and in the neighbourhood of elbow joint; Colles's fracture; Pott's fracture.

Begin at the end of the second week in fractures of the long bones.

Begin at the end of the third week in fractures of the femur.

PASSIVE MOVEMENTS.—Begin on the third day without disturbing splints, e.g., moving fingers in Colles's fracture.

As in massage when splints have to be removed, provided immobilization can be secured.

Complications arising during the Course of Treatment.

Hypostatic Pneumonia—Bedsores: In old people confined to bed.

Crutch Palsy, generally of the musculospiral.

Ischæmic Paralysis, or Volkmann's contracture. (*Vide* p. 115.)

Gangrene, caused by: Immediate injury to the soft parts—Subsequent thrombosis of the vessels—Rupture of the Vessels—Septic inflammation in a compound fracture—Bandage applied too tightly—Swelling of a part under a bandage—Flexion of a limb after bandaging.

Sloughing, from pressure of a badly-padded splint.

Compound Fractures.—Owe all their special features to:—

SEPTIC INFECTION through the wound. If this does not occur, or can be removed, the case progresses and is treated like a simple fracture.

COMPLICATIONS and results in septic cases: Necrosis of part of the bones—Osteomyelitis—Septicæmia or pyæmia—Secondary hæmorrhage.

PROCESS OF REPAIR IN SEPTIC CASES.—The inflammatory changes, instead of being plastic, are destructive in character. Pus is produced instead of granulation tissue. Bones in infected area have their vessels thrombosed by septic clot, and hence necrose. Necrosed bone is cast off. Wound and gap between bones fill with granulation. The granulations calcify and then ossify.

Time taken by repair is two to eight weeks longer than in aseptic cases, as this time is required to separate the necrosed sequestra.

Treatment of Compound Fractures.

Anæsthetize. Disinfect as thoroughly as possible.

Especially do this to any protruding fragment before reduction.

Remove crushed tags of skin and comminuted bone fragments.

Wire the fragments when retention of position is difficult otherwise.

Drain wound with gauze or tube. Place in splints.

Non-Union of Fractures may be of four degrees:

(1) Delayed union; (2) Absolute non-union; (3) Fibrous union; (4) False joint.

COMMONEST SITUATIONS.—Bony processes giving muscular attachments (patella, olecranon, coracoid process, os calcis)—Humerus—Femur (especially the neck)—Tibia.

CAUSES.

WIDE SEPARATION OF THE FRAGMENTS (e.g., olecranon).

CONSTANT MOVEMENT (e.g., humerus).

INTERPOSITION OF SOFT PARTS (e.g., patella).

DEFECTIVE BLOOD SUPPLY (e.g., head of femur).

GENERAL BONE DISEASE—osteomalacia.

LOCAL BONE DISEASE: Malignant tumour—Gumma—Tubercle.

SCURVY OR GENERAL ASTHENIA, very rare.

N.B.—In senility, rickets, fragilitas, paralysis, tabes, bones break easily but heal readily.

IN CHILDREN AND YOUNG ADULTS the cause is very obscure—possibly an undue formation of cartilage instead of bone from the callus.

SIGNS OF NON-UNION.—Unnatural mobility—Pain: especially in fibrous union.

TREATMENT OF UNUNITED FRACTURES.—Continued *immobilization* in plaster-of-Paris, allowing limb to be used when *this can be* done without increasing displacement.

Rubbing bony ends together.
Passive hyperæmia.
Open operation: Refresh bony ends and fix by plates, wire, or screws.
Failing these: Retentive apparatus.

FRACTURES OF SPECIAL BONES.

INFERIOR MAXILLA.

CAUSES.—Direct violence (common), indirect (rare).

POSITION OF FRACTURE.—Between canine tooth and mental foramen—Behind angle of jaw—Coronoid process (rare)—Condyle (rare).

SIGNS.—Crepitus—Irregularity of line of teeth—Wound and bleeding into mouth—Generally compound by communication with mouth—Displacement: Anterior fragment downward, posterior fragment upward.
Of Condyle: This process is drawn forwards.
Of Coronoid process: It is drawn upwards.

COMPLICATIONS.—In compound cases: Necrosis—Septic pneumonia—Pyæmia.

TREATMENT.

1. Four-tailed bandage
2. Moulded leather splint } where displacement is slight.
3. Hammond's wire splint round the teeth.
4. Kingsley's splint: Mould over teeth—Connected with outside metal bars—Bars bandaged to chin.
5. Moulded splint over teeth fixed by bars and screws to moulded splint outside and below jaw.
6. Splint between upper and lower jaw, the mouth being open. Jaws bandaged together.
7. Wiring fragments.

Immobilization is required for three weeks. Feed patient between cheek and teeth. Use frequent antiseptic mouth-washes.

RIBS.

CAUSE.—Indirect violence (commonest). Ribs break near the angle. Direct violence.

RIBS AFFECTED.—Fifth and eighth most commonly.

SYMPTOMS.—Pain on breathing—Irregularity and crepitus—Pain over [illegible]re on pressing sternum to spine.

COM[illegible]NS (especially in cases of direct violence)—Pleurisy [illegible]sion: Serous, purulent, blood or air—Wound of [illegible]ptysis—Surgical emphysema.

[illegible]Strapping rather more than half the chest [illegible]ion of expiration. Firm bandage. [illegible]c complications exist—Rest in bed.

CLAVICLE.

CAUSES.—Indirect violence by falls on the hand—Direct violence (rare).

FOUR POSITIONS :—

1. STERNAL END (rare) : Little displacement—Inner end drawn up.
2. MIDDLE (common situation) : Between rhomboid and coraco-clavicular ligaments—Fracture runs backwards and inwards —Inner end raised—Outer end depressed, tilted down, approximated to mid-line—Head bent over to affected side — Shoulder displaced downwards, inwards, and forwards.
3. BETWEEN CONOID AND TRAPEZOID LIGAMENTS (direct violence) : Slight displacement—Contusion and crepitus.
4. AT ACROMIAL END (direct violence) : Outer end, downwards and forwards.

VARIETIES AND COMPLICATIONS. — Greenstick fracture (common in children)—Compound (rare)—Injury to subclavian vein (rare)—Injury and thrombosis of subclavian artery : gangrene —Wound of pleura and lung.

TREATMENT.

REPLACEMENT.—Pull shoulder backwards and upwards. Manipulate fragments.

RETENTION. Required for four weeks.

1. REST IN BED. Pillow between scapulæ. Arm bandaged to side.

   Callus formation reduced to a minimum. Hence useful for ladies.
2. SAYRE'S METHOD. (routine method).—Strapping round arm and then round chest, keeping shoulder back. Strapping over opposite shoulder and under bad elbow, keeping shoulder up.
3. BANDAGING both shoulders back. Supporting arm in a sling.

SCAPULA.

VARIETIES.

ACROMION. CORACOID PROCESS. BODY.	Result from direct violence. Cause slight deformity or crepitus. Treat by raising and fixing the arm.

NECK.—Anatomical neck. Rare complication of a dislocation.

Surgical neck. Fracture runs downwards from the supra-scapular notch. The separated piece includes the coracoid process.

SIGNS. Flattened shoulder—Prominent acromion—Lengthened arm.

TREATMENT. Axillary pad—U-shaped leather splint for shoulder.

HUMERUS.

Anatomical Neck.—" Intracapsular fracture." Results from direct violence.

SIGNS. Loss of mobility. Great bruising. Head feels irregular from the axilla. Detached fragment may be felt loose in the capsule. Slight shortening of the arm. Crepitus is difficult to obtain. Impaction drives upper fragment into the lower. Any alteration in shape of shoulder is obscured by swelling.

COMPLICATIONS.—Non-union or mal-union may occur. Massive callus is formed during repair. Great stiffness of the joint usually results.

TREATMENT.—Support the arm in a sling. Early massage, and movements from the third day. Open operation to remove or peg an ununited fragment. Never disimpact an impacted fracture.

Surgical Neck.—" Extracapsular fracture."

CAUSES.—Direct, rarely indirect violence.

LINE.—Below the tuberosities. Between the attachments of the short rotators and the attachments of the pectoral, teres major, and latissimus.

DISPLACEMENT.—Upper fragment rotated outwards and adducted by the short rotator muscles. Lower fragment raised by deltoid, biceps, triceps, etc., drawn inwards by the pectoralis major, teres major, and latissimus.

SIGNS.—Shoulder itself is unaltered in outline. Depression occurs below the shoulder. Line of the humerus points upwards and inwards to axilla. Crepitus is well marked on rotation. Marked shortening occurs. Upper end of the lower fragment may be felt in axilla.

IMPACTION may occur. Lower fragment is driven into the upper. Diagnosis can then only be made certain by X rays.

COMPLICATIONS.—Injury to the axillary vessels. Painful pressure on, or injury of, the nerves, especially of the circumflex nerve. Dislocation may co-exist.

TREATMENT.

1. Reduction by extension and manipulation, under an anæsthetic in severe cases. The result should always be tested by a skiagram.
2. Axillary pad, bandage, and sling.
3. Middeldorpf's triangular axillary splint.
4. Open operation for complicated cases.

Massage from the first week. Manipulations from the second week. Never disimpact an impacted fracture.

Separation of the Upper Epiphysis.—Occurs up to about twenty. End of diaphysis is conical, and the epiphysis is hollowed out. Displacement is usually incomplete owing to the periosteal sleeve. Upper end of diaphysis projects forwards and inwards.

TREATMENT.—As for the last. Open operation if reduction cannot be performed.

Great Tuberosity.

CAUSE.—Direct or muscular violence.

DISPLACEMENT.—Upwards and backwards by supra- and infraspinati.

SIGNS.—Bruising and widening of the shoulder—Marked crepitus.

TREATMENT.—Pegging through an open operation. Early massage and movements.

Shaft.

CAUSES. Direct, indirect, or muscular violence.

DISPLACEMENT.

1. Below the pectoral insertion and above the deltoid: Upper fragment inwards, lower fragment upwards and outwards.
2. Below the deltoid insertion: Upper fragment outwards, lower inwards and upwards.
3. In lower third: Lower fragment upwards in front of or behind the upper.

COMPLICATIONS.

Injury of the musculospiral nerve or its involvement in callus.

Non-union, due to: Interposition of fibres of the triceps muscle or defective immobilization.

TREATMENT.

1. Internal angular splint from axilla to wrist, with three short splints over the humerus.
2. Middeldorpf's triangle.

Immobilization continued for five weeks.

Lower End.

1. TRANSVERSE SUPRACONDYLOID.

Causes.—Indirect violence by falling on the hand with a bent elbow. Direct violence by falling on the bent elbow.

Displacement.—Lower fragment upwards in front of or behind the upper.

Signs.—Shortening of the arm from acromion to condyle. Increase in thickness of the lower end of the humerus from before backwards. Unaltered relation of condyles to olecranon. Apparent shortening or lengthening of the forearm according to backward or forward displacement.

Treatment.

(*a*) For anterior displacement: Anterior angular splint to the elbow. Posterior straight splint to the humerus.

(*b*) For posterior displacement: Posterior angular splint to the elbow. Anterior straight splint to the humerus.

2. SEPARATION OF THE LOWER EPIPHYSIS.—May occur up to fourteen or fifteen. Common in children. After fourteen or fifteen it does not involve the internal condyle. Displacement is backwards and outwards.

Treatment.—Reduce by flexion. Fix in position of flexion by splints or bandage. Sling for the forearm.

3. CONDYLES.

INTERNAL CONDYLE. Direct violence :—

(*a*) Intracapsular. Involves the epicondyle and part of the trochlea. May result in cubitus varus.

(*b*) Extracapsular. Involves the epicondyle only.

EXTERNAL CONDYLE. Always intracapsular. Involves the epicondyle and part of the capitellum. May result in cubitus valgus.

SIGNS.—Great swelling and ecchymosis of the joint. Crepitus on grasping the affected condyle. The extended elbow can be adducted or abducted.

TREATMENT.—Fix in a position of flexion by an anterior angular splint or bandages. Employ massage and movements from the end of the first week. If stiffness results in spite of this, excise the displaced fragment. If X rays show that reduction is from the first unsatisfactory, then open the part and fix the condyle by peg or wire.

4. T-SHAPED FRACTURE INTO THE JOINT.—Rapid effusion of blood into the joint. Crepitus on moving the condyles on one another.

TREATMENT as above. The open operation is specially indicated.

ULNA.

Olecranon.

CAUSES.—Falls on the elbow, or muscular action (rare).

DISPLACEMENT.—Drawn upwards and backwards by the triceps.

SIGNS.—Mobility, with crepitus of the fragment. Great distention of the joint.

COMPLICATIONS.—Forward dislocation of the arm at the elbow. Injury of the ulnar nerve. Fibrous union.

TREATMENT.

1. Open operation and wiring.
2. If an operation is refused, straight anterior splint and 8-bandage.

Coronoid.—Occurs as a complication of backward dislocation of elbow. The dislocation is easily reduced, is accompanied by crepitus, and easily recurs.

TREATMENT.—Fixing in acute flexion. Fixing in posterior angular splint. Using early massage and movements (end of first week).

Shaft.

CAUSE.—Direct violence.

COMPLICATION.—Dislocation of the radius.

DISPLACEMENT.—Lower fragment towards the radius (pronator quadratus), upper fragment forwards (brachialis anticus).

TREATMENT.—As in fractures of both bones.

Styloid Process.—May complicate Colles's fracture of the radius.

RADIUS.

Head.—May be broken as part of a comminuted fracture of elbow. May require excision for non-union or mal-union.

Neck.—Fracture between the head and bicipital tuberosity.

DISPLACEMENT.—Lower fragment forwards (biceps).

SIGNS.—Upper end of the lower fragments forwards. Loss of voluntary rotation.

TREATMENT.—Posterior angular splint. Pad in front of the upper end of the radius.

Shaft.

1. ABOVE PRONATOR TERES INSERTION.
   Lower fragment fully pronated and drawn inwards (both pronators).
   Upper fragment fully supinated (biceps and supinator brevis).
   TREATMENT.—Posterior angular splint. Arm fixed in full supination.
2. BELOW PRONATOR TERES INSERTION.
   Upper fragment drawn forwards and inwards (biceps and pronator) in position between supination (biceps) and pronation (pronator).
   Lower fragment inwards by the pronator quadratus.
   TREATMENT.—Anterior and posterior arm-splints. Hand fully adducted and midway between pronation and supination.

Lower End, Backward Displacement (Colles's Fracture).

CAUSES, ETC.—Falls on the outstretched palm. Common in elderly women.

POSITION.—Within one inch of the joint.

DIRECTION.—Transverse, or, more commonly, oblique from above, downwards, forwards, and inwards.

DISPLACEMENT.—Is of a quadruple nature. The lower fragment is displaced:

1. Upwards towards the elbow.
2. Backwards by the direction of the blow and direction of the limb.
3. Rotated round the ulna styloid as a centre, so that the outer part is more displaced than the inner.
4. Rotated backwards round the line of fracture, so that the back of the fragment is more displaced than the front.

DEFORMITY.—Is also of a fourfold character :—

1. Radial styloid is higher than normal. i.e., it is on a level with the ulna styloid instead of being below it.
2. Hand is displaced backwards, and there is a bony prominence at the back of the wrist, with another bony prominence at a higher level in front of the wrist (dinner-fork deformity).
3. Hand is abducted to the radial side, with marked prominence of the ulna styloid process.
4. Wrist is rotated backwards, so that the joint surface looks backwards as well as downwards.

IMPACTION is usually well marked. Hence crepitus and mobility will be absent and the fracture may be mistaken for a sprain. The relative position of the styloid processes is the most important point in guarding against this.

The posterior part of the shaft is driven into the lower fragment.

The anterior part of the fragment is driven into the shaft.

TREATMENT.—Reduction of the deformity is absolutely essential. Disimpaction must be carried out under anæsthetic.

Fix on Carr's splint, or any splint which maintains efficient adduction of the hand and leaves fingers free.

Allow finger movements from third day.

Remove splints in the third week.

Lower End, Forward Displacement (Smith's Fracture).

CAUSED by falls on the back of the flexed wrist.

Similar to a Colles's, but all the posterior displacements are now anterior. Produces a rather more square angle, which has given it the name of "the gardener's fork."

Separated Lower Epiphysis.—Occurs up to twenty.

Similar to a Colles's fracture. The displacement is more marked. Impaction is rare.

Treatment is the same.

Both Ulna and Radius.

CAUSES.—Direct or indirect violence.

POSITION.—Radius is usually broken higher up than the ulna.

DISPLACEMENT.—Broken ends tend to be drawn together by the pronators and supinators.

TREATMENT.—Place in full supination or midway between pronation and supination. Fix between two splints. Arrange an interosseous pad to separate the bones. Be careful to have the splints wider than the limb, so that the bandages do not press the bones together.

COMPLICATION.—Synostosis of radius and ulna.

METACARPALS AND PHALANGES.

CAUSE.—Direct injury. Either transverse or longitudinal fissured fractures. Often run into joints and complicate sprains. Should always be looked for by X rays.

TREATMENT.—Any lateral deviation at the joints should be corrected. Immobilize by finger splints. Early massage and movements.

PELVIS.

Above the Brim of the True Pelvis.—Parts of the iliac crest or the crest of the pubis (rare) may be broken off by direct violence.

TREAT by rest in bed, with pelvic bandage.

Involving the True Pelvic Cavity.

CAUSES.—Crushes or being run over.

POSITION.—Through the pubic and ischial rami into the obturator foramen. Also secondarily through the sacro-iliac joint of the opposite or same side.

COMPLICATIONS.—Laceration of the urethra, bladder, vagina, or rectum.

TREATMENT.—Rest in bed for six or eight weeks. Poroplastic pelvic belt. Appropriate treatment for visceral complications. Tie in a catheter for some days if there is hæmaturia or retention of urine.

Acetabulum.

POSTERIOR AND UPPER LIPS, with dorsal dislocation of hip.

REDUCE and treat by extension and extreme abduction to prevent recurrence.

FLOOR OF THE ACETABULUM, the femoral head being driven into the pelvis. Head of the femur can be felt per rectum.

REDUCE under an anæsthetic if possible.

Tuber Ischii, Sacrum, Coccyx.—These fractures require no special description. A fractured coccyx may lead to great pain, or unite in a position in which it encroaches on the pelvis; in either case it is best excised.

FEMUR.

At the Junction of the Head and Neck—"Intracapsular fracture."

CAUSES.—Slight violence of an indirect nature. Occurs in old people, generally in old women. Atrophy of the bony neck, and a more horizontal position of the neck than normal, predispose to its occurrence.

ANATOMY.—Transverse or oblique cleavage between the head and neck. A few fibres of the capsule may feebly connect the two. The majority of the nutrient vessels of the head which run up from the neck are torn.

Impaction is rare. The neck is driven into the head.

DISPLACEMENT.—Rotated outwards by weight of limb and external rotation. Drawn up by hamstrings and quadriceps. But the capsule prevents much upward displacement. The actual shortening is therefore slight—about one inch.

REPAIR.—Usually defective. Non-union or fibrous union due to: (1) Bad vascular supply of the head. (2) Danger of keeping the patient in bed. (3) Difficulty in fixing the fragments in apposition.

TREATMENT.—Rest in bed with the leg between sand-bags. Weight extension.

If any signs of congestion of the lungs appear, the patient must get up and walk on crutches.

Never disimpact, and always examine such cases very gently for fear of disimpaction.

At the Junction of the Neck with the Trochanters—" Extracapsular fracture."

CAUSES.—Great violence directly applied to the trochanter major. Usually occurs in adult men.

ANATOMY.—Cleavage is chiefly in the intertrochanteric line. Comminution is common. The neck, great trochanter, and shaft are the chief fragments. Impaction is the rule; the neck is driven into the great trochanter. The joint is always involved in front, though the fracture is usually "extracapsular" behind.

DISPLACEMENT.—Leg is rotated outwards by its own weight and the predominence of the external rotators.

SHORTENING IS WELL MARKED, and may be two inches. The length from the anterior superior iliac spine to the malleolus is shortened. The trochanter is raised above Nélaton's line. The horizontal side of Bryant's triangle is shortened.

The trochanter is often nearer to the mid-line than is normal, "the bi-trochanteric test."

OTHER SIGNS.—Crepitus is absent when impaction exists. Marked bruising about the trochanter. The trochanteric region is thickened. The ilio-tibial band is relaxed. The trochanter rotates round the arc of a smaller circle than is normal (if not impacted).

TREATMENT.—Always disimpact in young healthy patients where there is much displacement.

Long Liston's splint from the axilla to below the ankle, maintained from rotating by transverse bar.

Limb is pulled on by weight attached by strapping and bandages well above the knee.

Hodgen's splint is often more effectual than Liston's in reducing the deformity.

Maintain splint and extension for six weeks.

Fracture of the Great Trochanter alone.

Fracture of the Shaft just below the Trochanter.

Rare injuries which do not require any special description.

Separation of the Upper Epiphysis.—The head does not join the shaft until eighteen. Its separation results from falls on the leg or hip. Occurs in patients of about fourteen. Its signs are very vague at the time. It results in marked coxa vara from the resulting reconstruction of the head of the bone (p. 123).

Shaft.

CAUSES.—Direct or indirect violence.

POSITION.—Upper, middle, or lower thirds.

DIRECTION.—Oblique or spiral.

DISPLACEMENT.—The lower fragment is always drawn up by the thigh muscles, and everted by weight of the limb.

Shaft of the Femur, Displacement, *continued.*

In Fracture of the Upper Third, the upper piece is flexed by the psoas; the lower piece is adducted.

In Fracture of the Lower Third, the lower fragment is flexed at the knee by the gastrocnemius; the upper fragment is driven down so as to penetrate the knee joint or the quadriceps muscle and skin.

TREATMENT.—When there is not much forward or backward displacement: A long Liston with short splints round the fracture. Weight extension.

When marked forward or backward tilting occurs: Hodgen's splint. Tenotomy of the tendo Achillis helps to release the flexed lower fragment.

When forward or backward displacement in the lower third occurs without much shortening: A double inclined plane or M'Intyre's splint.

In fractures of the lower third where the upper fragment has penetrated the joint or the quadriceps: Open operation.

In small children: Slinging the limb up at right angles to the body, which acts as an extending weight.

Lower End.

TRANSVERSE FRACTURE.—Similar to that of the lower third of the shaft. Special liability to backward tilting of the lower fragment by the gastrocnemius. This may cause an injury of the popliteal vessels.

T-SHAPED IN THE JOINT.

SEPARATION OF ONE CONDYLE.—Rare fractures. Crepitus on pressing one or both condyles. Marked effusion into the joint.

SEPARATION OF THE LOWER EPIPHYSIS.—Epiphysis is displaced forwards. The lower end of the shaft presses on the popliteal vessels.

TREATMENT OF FRACTURES OF THE LOWER END.—Open operation when the joint is involved and much displacement occurs.

Hodgen's or M'Intyre's splint in other cases.

Massage and passive movement in the third week.

PATELLA.

CAUSES.

Direct Violence.—Usually produces a stellate or fissured fracture. The aponeurosis is not separated. Little or no displacement results.

Indirect, i.e., muscular violence of the quadriceps extensor. This is the common form to which the following applies. The patella is broken across the femoral condyles by force applied to its two ends.

ANATOMY.—Marked displacement of the fragments. The ragged aponeurosis of the quadriceps hangs over the divided bony ends. Hæmorrhage and effusion into the joint.

SIGNS.—Inability to extend the knee. Presence of the upper fragment high above the knee. Gap between the fragments.

UNION.—By fibrous tissue forming a band between the fragments. Absence of bony union is due to (*a*) Interposition of aponeurosis between the fragments; (*b*) Muscular traction separating and tilting the fragments.

TREATMENT.—When separation of the fragments exists, open the joint at the end of the first week. Clear out the blood-clot and cut off the tags. Unite the fragments by wire, which must not encroach on the cartilage.

(All methods of approximating the fragments without freely opening the joint are liable to fail owing to the interposition of the aponeurotic tags.)

Passive movement at the end of first week.

Active movements at the end of the second.

TIBIA AND FIBULA.

Tibia.

AT UPPER END.—Generally results from direct violence. Sometimes T-shaped into the joint. Not much displacement.

TREATMENT.—M'Intyre's splint.

OF THE TUBERCLE.—This may ossify from a separate nucleus, which appears about twelve and joins shaft at fourteen. Generally it grows down as a beak from the head. Generally detached by muscular action of the quadriceps. Sometimes by direct violence.

TREAT by plaster in position of extension, or wire if there is much separation.

OF THE SHAFT.—Caused by direct blows. Transverse above and oblique below. Irregularity of anterior border. Pointed end of upper fragment may pierce the skin.

TREAT by back or side splints, and then plaster.

OF THE INTERNAL MALLEOLUS.—From a blow on the ankle, or from a wrench of the foot inwards. Not so much displacement as in Pott's. Likely to be followed by a stiff ankle.

TREAT by side splints or wiring.

Fibula.

SIGNS are: (1) Localized pain on pressing the two bones together; (2) Loss of the fibula spring.

TREAT by plaster immobilization.

Tibia and Fibula.

WHEN FROM DIRECT VIOLENCE.—Bones are broken at the same level. Fracture is transverse. Not much displacement. Great contusion.

Tibia and Fibula, *continued.*

WHEN FROM INDIRECT VIOLENCE.—Tibia breaks at junction of middle and lower third. Fibula breaks higher up. Fracture is oblique downwards, forwards, and inwards. Great displacement. Lower fragment drawn up and rotated outwards. Upper fragment often pierces the skin.

Spiral shaped fracture is produced by torsion. Is specially difficult to reduce.

TREATMENT.

REDUCTION is often difficult. Traction under an anæsthetic with knee flexed. Division of tendo Achillis may help.

PUT UP: (1). In two side splints, the outer having a foot-piece; or (2) In back splint and two side splints. Attach extension weight to lower fragment, and sling from cradle when persistent shortening occurs.

Correct rotation so that the inner margin of great toe, inner malleolus, and inner margin of the patella are in a line.

At the end of three weeks use a plaster or leather splint in two halves, and massage daily.

OPEN OPERATION AND WIRING THE FRAGMENTS.—In cases where reduction otherwise is impossible. In cases where reduction cannot be maintained. Especially in the case of working men.

Reduces the time of recovery. Enables massage and passive movement to be performed much earlier.

Pott's Fracture, or fracture-dislocation of the ankle joint.

CAUSED BY indirect violence—Tripping or turning the foot.

DISPLACEMENT.—Foot displaced and rotated: (1) Outwards (far the commonest); or (2) Inwards, (3) Backwards, or (4) Upwards (rare).

ANATOMY.—The fibula is always broken three or four inches above the ankle. The other lesions affect the ligaments and tibia in four different ways.

1. Simple rupture of the internal lateral ligament.
2. Fracture of the internal malleolus.
3. Either of the above (1) or (2), combined with rupture of the interosseous ligament and great outward or even upward dislocation of the foot (Dupuytren's fracture).
4. Fracture of the lower end of the tibia. Lower end of the upper tibial fragment forms sharp prominence which may project through the skin.

SIGNS.—Great swelling from bleeding and effusion into the ankle joint. Foot is displaced, generally outwards and slightly backwards, and rotated outwards.

Internal malleolus is very prominent. Fracture of the fibula three or four inches above ankle.

Crepitus may be felt, and there is loss of the fibula spring.

TREATMENT.

REDUCE UNDER ANÆSTHETIC. Traction with rotation of the foot. Keep the knee bent to relax the gastrocnemius. Cut tendo Achillis where much spasm exists.

PUT UP on back splint with foot-piece and two side splints. Inner splint with thick pad to come over inner ankle (Dupuytren's splint).

POSITION TO BE ATTAINED.—Foot at right angles to the leg. Heel brought forward. Rotation and external displacement corrected until great toe, inner ankle, and patella are in line.

In cases where satisfactory reduction is impossible, open operation and wiring or pegging fragments.

AFTER-TREATMENT.—Remove side splints and give daily massage at the end of the first week. Gentle passive movements at the end of a fortnight. In three weeks provide case splint, which can be daily removed for passive exercise and massage.

CHAPTER XIX.

DISEASES OF BONES.

INFLAMMATORY DISEASES OF BONES.

Structure of Bones.

HARD PARTS.

- DENSE BONE forms shaft of long bones; outer and inner tables of flat bones and outer shell of short bones.
- CANCELLOUS BONE forms interior of epiphyses and epiphyseal ends of shafts of long bones, also interior of short and flat bones.

SOFT PARTS.

- PERIOSTEUM.—A vascular membrane outside bones, with deep layer of growing cells. It ceases where the epiphysis joins the shaft. From it growth in thickness of shaft occurs.
- MEDULLA.—A very vascular and lymphatic mass of cellular tissue inside the cancellous tissue and in shaft of long bones.
- HAVERSIAN CANAL SYSTEM.—Minute vessels which traverse the dense bone and unite periosteal with medullary vessels.
- CARTILAGE.
    - Epiphyseal. A vascular layer of growing cartilage between epiphysis and diaphysis, from which the growth of the shaft in length takes place. Its circumference is connected with the periosteum.
    - Articular. Smooth cartilage covering articular ends of the diaphysis. Non-vascular.

VESSELS.

- NUTRIENT ARTERY, with vein and lymphatics, pierce shaft and end in the medulla.
- PERIOSTEAL VESSELS supply the periosteum and send inward branches to Haversian canals.
- EPIPHYSEAL VESSELS, given off from the vascular anastomosis round the joint to the epiphysis, especially at the junction with the shaft, where they supply the epiphyseal cartilage.
- HAVERSIAN VESSELS.—Minute twigs which join the periosteal with the medullary vascular system.
- LYMPHATICS are present in the perivascular sheaths.

Relation of Inflammatory Processes to Bone Tissues.

ORIGIN.—Inflammation always begins in the vascular soft tissues, i.e., in periosteum, medulla, or epiphyseal cartilage.

EXTENSION.—Inflammatory processes spread from periosteum to medulla or from medulla to periosteum by (1) Haversian canal system ; (2) Epiphyseal cartilage.

THE HARD PARTS of the bone may be regarded as intercellular material, which will react in three different ways to inflammation.

1. NECROSIS.—Death *en masse* from acute inflammation with vascular thrombosis.
2. CARIES.—Molecular necrosis or ulceration, from sub-acute inflammation.
3. SCLEROSIS from chronic inflammation laying down new bone.

Necrosis is caused by :

Periostitis : Acute localized — Acute infective — Chronic syphilitic.

Osteomyelitis : Acute localized or septic—Acute infective—Subacute septic, tuberculous, syphilitic.

Toxic poisoning : Phosphorus—Mercury.

Senile necrosis corresponding to senile gangrene.

" Quiet necrosis " is a traumatic form.

NECROSIS corresponds to gangrene or sloughing of soft parts.

Caries.—An inflammatory rarefaction of bone :—Simple (as in repair of fractures)—Septic (inflammation of cancellous tissue)—Tuberculous (the commonest form of caries)—Syphilitic.

It corresponds to ulceration of soft parts, and the fibroblasts of granulation tissue are represented by multinuclear cells called osteoclasts, which eat into and absorb the cancellous bone.

CARIES SICCA.—Caries without suppuration.

CARIES SUPPURATIVA.—Caries with suppuration.

CARIES FUNGOSA.—Caries with exuberant granulations.

CARIES NECROTICA.—Caries associated with necrosis of minute portions of bone: corresponding to ulceration with sloughing of soft parts.

Sclerosis results from chronic inflammatory processes.—Chronic periostitis—Chronic osteomyelitis.

Simple, in the proximity of some inflammatory focus in soft parts, e.g., chronic ulcer over tibia.

Syphilitic—much the commonest.

Tuberculous—rare, and occurs beyond the area of tuberculous affection.

It corresponds to fibrosis of soft tissue. and results from a laying down of fresh bone (corresponding to intercellular fibrous tissue) by medulla, periosteum, and Haversian vessels.

Periostitis.

VARIETIES.

ACUTE LOCALIZED.

ACUTE DIFFUSE. Generally associated with infective osteomyelitis as cause or effect.

CHRONIC : Simple—Tuberculous—Syphilitic (local, general).

Acute Local Periostitis.

CAUSES.—Traumatism—Rheumatism—Specific fevers—Pyæmia—Gout—Alveolar abscess.

RESULTS.—(1) Resolution; (2) Chronic thickening; (3) Suppuration with necrosis.

PATHOLOGY.

IN ASEPTIC CASES.—Exudation from deep layer of periosteum. Organization of this exudation.

IN SEPTIC CASES.—Exudation from deep layer of periosteum. Thrombosis of vessels. Suppuration of the exudation. Rupture of vessels passing from periosteum to underlying bone, caused by tension of exudation. Necrosis of fragment of bone which is separated from its periosteum.

Necrosed fragment is called the SEQUESTRUM.

Living bone round sequestrum is rarefied by the inflammatory proliferation of cells forming GRANULATION TISSUE.

REACTION BETWEEN GRANULATION TISSUE AND SEQUESTRUM may be:

1. Absorption of sequestrum if small and sepsis is at an end.
2. Erosion of sequestrum by granulations until it is separated from living bone.
3. If sepsis is still active, the living bone will be absorbed by granulation tissue until sequestrum lies free. Occupies about four to eight weeks.

Deep surface of separated periosteum in the meantime forms new bone—the INVOLUCRUM.

Pus under the periosteum escapes through periosteum and soft tissues. This opening remains in the involucrum, and is called a CLOACA.

The sequestrum escapes through the cloaca when it is loose and if the cloaca is large enough.

After the escape of the sequestrum, the granulation tissue lining the living bone becomes ossified, forming a dense layer of osteosclerosis.

Acute Diffuse or Infective Periostitis is practically the same disease as acute osteomyelitis, the one being the cause or effect of the other.

Chronic Periostitis is described under the head of "Chronic Osteoperiostitis."

Osteomyelitis.

VARIETIES.

ACUTE INFECTIVE—associated as cause or effect with infective periostitis.

ACUTE SEPTIC OR TRAUMATIC.

SUBACUTE: Simple or infective—Associated with repair of fractures, and separation of sequestra.

CHRONIC: Simple—Tuberculous—Syphilitic.

EPIPHYSITIS is an acute infective osteomyelitis of the epiphyses.

Acute Infective Osteomyelitis.

ETIOLOGY AND PATHOLOGY.

- CHILDREN, five to fifteen being the commonest age, that is, when the epiphyseal cartilages are in full activity.
- Supervenes on a condition of general DEBILITY.
- Often follows EXANTHEMATA, e.g., scarlet fever.
- Often follows SEPTIC ULCERS of mouth, throat, or intestines.
- Generally results from a BLOW or WRENCH.
- Caused by entrance of PYOGENIC MICRO-ORGANISMS:
    - *Staphylococcus pyogenes aureus.* Common type.
    - *Staphylococcus pyogenes albus.* Mild type.
    - *Streptococcus pyogenes.* Young children. Very fatal. Less extensive necrosis.
    - *Pneumococcus.*
    - *Bacillus coli commune,* in mixed infection.
- PRIMARY BONY FOCUS is usually the growing vascular tissue on shaft side of epiphysis.
- MAY BEGIN IN EPIPHYSIS—acute epiphysitis.
- MAY BEGIN AS LOCAL PERIOSTITIS, especially at the back of femur in young adults.

VARIETIES of infective osteomyelitis:—

1. LOCAL PERIOSTITIS.—Where the focus is near periphery the inflammatory process spreads outwards, and the case resembles acute local periostitis. Especially common at the back of the lower end of the femur.
2. DIFFUSE INFECTIVE PERIOSTITIS.—Suppurative inflammation may strip periosteum off the whole shaft, which then lies in an abscess cavity.
3. MEDULLARY OSTEOMYELITIS.—Medulla becomes inflamed, veins thrombosed, and pyæmia often follows. Haversian vessels are infected. Bony walls of Haversian canals being unyielding, the exudation causes strangulation of vessels. This cessation of circulation and the toxins kill the bone, producing total necrosis. Periosteum is infected. Its deep layers suppurate, and it is stripped off the shaft.
    - EPIPHYSES ESCAPE because
        - (*a*) They have an independent blood supply.
        - (*b*) They are separated from the shaft by comparatively avascular zone of cartilage.
        - (*c*) They have no periosteum through which infection can take place.
4. ACUTE ARTHRITIS.—Suppuration will extend into the neighbouring joint:
    - (*a*) If the epiphyseal cartilage lies within the joint capsule, e.g., hip or elbow.
    - (*b*) In infants where epiphysis consists of thin cartilage only: this may be perforated, and pus so reaches the joint cavity.
    - (*c*) By extension along soft tissues, e.g., biceps tendon into the shoulder joint.

Acute Infective Osteomyelitis, *continued.*

5. CHRONIC ABSCESS.—Where infection is mild and resistance is good, the inflammatory process may be limited, and an abscess forms in the cancellous tissue, surrounded by dense thickened layer of osteosclerosis. Commonest at upper end of tibia, lower end of femur, or lower end of tibia. A similar abscess may result from tubercle.

TERMINATION.—Apart from complications, all varieties except the last two present three stages:—

1. STAGE OF INFECTION and suppuration, when the periosteum is stripped off all or a part of the shaft.
2. STAGE OF PERIOSTEAL OSSIFICATION. The pus escapes through one or more openings in the periosteum and soft tissues. These are the cloacæ. The deep layers of periosteum produce a shaft of new bone—the involucrum.
3. STAGE OF SEPARATION of the sequestrum. The sequestrum is separated from the living bone (in total necrosis this will be at its two ends) exactly as described under local acute periostitis. This occupies about four weeks in the case of small bones, up to three months in the case of the femur.

SYMPTOMS.—Rigor, with high fever. Severe pains in the limb. Brawny swelling of soft parts. Absence of the scarlet flush of superficial inflammation. Tenderness is very great. Joint can often be moved gently without pain.

IN PERIOSTEAL CASES: Superficial fluctuating swelling.

IN MEDULLARY CASES: Severe toxic and pyæmic symptoms.

COMPLICATIONS:—

LOCAL.—Acute arthritis (see above). Sloughing of the periosteum, preventing the regeneration of the bone. Destruction of epiphyseal cartilage, stunting growth. Overgrowth of the bone by stimulation of chronic inflammation, and long-standing hyperæmia.

GENERAL.—Septicæmia—Pyæmia—Lardaceous disease.

PROGNOSIS.—Always grave; it is severe in proportion to: Youth of patient—Debility of patient—Size of the bone—Extent of the bone disease—Virulence of infection.

Medullary primary infection is worse than primary periosteal infection.

Streptococcal infection is almost hopeless.

Staphylococcus aureus infection is grave.

Staphylococcus albus is milder.

TREATMENT.

GENERAL TREATMENT suitable for fever.

CUT DOWN through the periosteum on to the shaft AS SOON AS THE DIAGNOSIS IS MADE OR APPEARS PROBABLE.

TREPHINE or gouge shaft so as to open into medulla or cancellous tissue.

SCRAPE AWAY soft material, and drain.

If total necrosis has occurred, cut sequestrum in two and twist each end off from epiphysis.

Except in case of humerus or femur, when sequestrotomy must be delayed until the involucrum has formed.

When involucrum has formed, wait until examination by the probe shows that the sequestrum is loose, then enlarge cloacæ and remove sequestrum.

AMPUTATION may be called for: When sequestrum is inaccessible e.g., at the back of lower end of the femur—When pyæmia is present—When exhaustion threatens life—When suppuration in a large joint occurs—When lardaceous disease has begun.

Acute Septic or Traumatic Osteomyelitis.

CAUSES.—Septic compound fractures—Amputations—Any bone operation.

SYMPTOMS.—Inflammation and suppuration of wound—End of the bone is seen or felt to be white and bare.

High temperature, with rigors or pyæmia, may occur.

Portion of the bone is extruded as the

SEQUESTRUM: Tubular or conical. Its outer surface is pitted by granulations.

TREATMENT.—Open up wound. Scrape out medullary tissue and drain. Amputate if pyæmia persists.

Chronic Osteoperiostitis.—Usually primarily a periostitis, but by extension the disease affects the Haversian canals and medulla, and produces a general sclerosis and thickening of the bone.

LOCAL.—Caused by local periostitis, e.g., beneath a chronic ulcer, or by trauma, rheumatism, syphilis. A hard node is formed by new periosteal bone, in which the Haversian canals run at right angles to the surface. Later this becomes sclerosed.

DIFFUSE.—As an extension from the local variety.

In syphilis as a primarily diffuse periostitis or round a central gumma.

In tubercle from a central abscess or other chronic focus.

SYMPTOMS.—Deep aching pain, worse at night. Bone is felt to be thickened, and seen to be so by the X-rays.

TREATMENT.—Rest, counter-irritation, with iodides internally.

If the pain is severe, an operation may be necessary. The bone is exposed, the periosteum stripped off, and the medulla opened by the trephine or gouge.

Amputation may be required in the worst cases.

TUBERCULOUS DISEASE OF BONES.*

Varieties of Tuberculous Disease.

1. MILIARY TUBERCLE.—Very rarely found in the bones in general infection.

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* Watson Cheyne, "Tuberculous Disease of Bones and Joints."

Varieties of Tuberculous Disease, *continued.*

2. CASEOUS FOCI.—Usually situated towards the end and near the surface. The following zones occur from within outwards :—
   - (*a*) A MASS OF CASEOUS MATERIAL occupies the centre. This represents the first seat of the disease, the cells of which have died and undergone fatty degeneration.
   - (*b*) GRANULATION TISSUE, containing tubercles in which all bony trabeculæ have been absorbed.
   - (*c*) A ZONE OF SCLEROSIS, containing small round cells instead of fatty tissue.
   - (*d*) A ZONE OF RAREFYING OSTEITIS at some distance from the deposit.

   The whole process spreads to the periphery, whether joint or periosteal surface.

3. NECROSIS WITH SEQUESTRUM FORMATION.—This also occurs chiefly in the ends of long bones. The sequestra vary in size, and are often wedge-shaped, with the base of the wedge towards the joint. Here three zones occur, viz. :
   - (*a*) THE CENTRAL SEQUESTRUM, in which the trabeculæ are thickened.
   - (*b*) A ZONE OF TUBERCULOUS GRANULATIONS.
   - (*c*) A ZONE OF RAREFYING OSTEITIS.

   The formation of caseous foci and of sequestra often occur at the same time and locality. The sequestra often remain attached for a long time to the living bone at one spot.

4. SUPERFICIAL CARIES.—This occurs beneath the cartilage of a joint or the periosteum of the shaft. The surface is covered by caseous débris. Beneath this is more or less osteosclerosis. At a little further distance a zone of osteoporosis. It very seldom extends deeper than a quarter of an inch from the surface.

5. TUBERCULOUS PERIOSTITIS.—Commonest in the ribs and vertebræ. Produces caries of the underlying bone, together with chronic abscesses spreading along the bones and tracking to a distance.

6. OSTEOMYELITIS (also epiphysitis).—Most often seen in the phalangeal, tarsal, and carpal bones.

   STRUMOUS DACTYLITIS is common in children. A localized swelling occurs over a phalanx or metacarpal bone, in which tubercular caseous processes expand the bone and break through the outer shell.

   In adults diffuse thickening from periostitis and the formation of central sequestra is commoner.

   In either case neighbouring structures are affected by extension, viz. : (1) Joints ; (2) Tendon sheaths ; (3) Adjacent bones, especially in the wrist and ankle.

7. CARIES SICCA.—Is very rare, except in the shoulder joint. Instead of soft tuberculous granulation tissue, firm fibrous tissue forms on the surface, and the bone steadily atrophies. *It is accompanied* by ankylosis with muscular atrophy.

8. DIFFUSE OSTEOSCLEROSIS.—This occurs for some distance along the shaft of a bone in the neighbourhood of an old tuberculous process. The bone becomes dense and thick.
9. DIFFUSE SOFTENING.—The rarest of all changes. Only seen in rapid cases where the disease spreads from one bone to another. There is a great enlargement of the medullary cavity and increase of the red marrow, with general absorption of the hard bone.
10. CHRONIC DEEP ABSCESS. — Usually situated near the epiphyseal line. It is lined by a thick pyogenic membrane and surrounded by a thickened mass of sclerosed bone. This thickening often extends up the shaft.

Distribution of Tuberculous Bone Lesions.

CRANIAL BONES. Rarely become carious with an external abscess.

VERTEBRÆ. Affected with central osteomyelitis or superficial caries.

RIBS. Periostitis with abscess.

LONG BONES:—Epiphyses: caseation, necrosis, osteomyelitis, or chronic abscess—this is much the commonest situation. Diaphyses: rarely are the seat of periostitis, osteomyelitis, or chronic abscess.

PELVIS. Near the sacro-iliac joint or any of the epiphyses, e.g., the crest of the ilium.

CARPAL, TARSAL, AND PHALANGEAL BONES. Commonly affected by osteomyelitis, necrosis, or caries. The os calcis, astragalus, scaphoid, or one phalanx, may be separately diseased, but usually more than one bone or joint are affected.

Clinical Signs.

PAIN. Generally in proportion to the depth of the lesion and its subjection to pressure.

SWELLING: (1) Of the bone; (2) Of the soft parts over it; (3) Abscess.

ABSCESS. An abscess forms without inflammatory phenomena, and in deep bone disease tracks to a distance.

FEVER. Usually absent before the abscess breaks or becomes infected. Occasionally is well marked in purely tuberculous abscess of bones.

JOINT SYMPTOMS, e.g., deformity, immobility, pain, etc.

Treatment.

CONSTITUTIONAL TREATMENT, with rest to the affected part by splints. Passive hyperæmia in suitable cases.

OPERATIVE.—When the above has failed, or on the occurrence of an abscess, the part is laid open, the diseased part scraped away, sequestra removed, and the cavity rubbed with iodoform or filled with iodoform wax.

AMPUTATION is rarely required, and then generally in the case of a finger, hand, or foot, where the disease has spread to several bones and joints.

SYPHILIS OF BONES.

Secondary Syphilis.

SYMMETRICAL LOCALIZED PERIOSTITIS forming hard nodes. These occur most commonly on the tibia, and are associated with aching pain, worse when the part is warm, e.g., at night. It is a late secondary lesion, and may also occur in the tertiary stage.

Tertiary Syphilis.

1. GUMMATA.—These are usually subperiosteal.

THE CRANIAL BONES are the commonest site, especially the frontals and parietals. A firm nodular swelling first appears, which then softens and fluctuates. The bone beneath presents (1) CARIOUS DESTRUCTION, whereby a circular clean-punched hole is produced which very rarely perforates the skull; (2) An OSTEOPLASTIC PERIOSTITIS at the margin, forming dense new bone; (3) AN EXTENSIVE SCLEROSIS, which may affect the whole vault of the skull or only that part under the gumma.

When the gumma breaks and sepsis is admitted, a FOUL ULCER results and the bone becomes necrosed and black.

BY AN EXTENSION OF THESE PROCESSES a large part of the cranium may be affected: (1) By worm-eaten erosion; (2) By heaping up of new bone; (3) By sclerosis; (4) By necrosis due either to sepsis or to a cutting off of the blood supply by sclerosis. The sequestrum remains for years without separating.

BY AN EXTENSION THROUGH THE SKULL, similar conditions may occur between the skull and dura, the deep surfaces of the bones being then affected.

THE PALATE AND MAXILLARY BONES, the vomer, ethmoid, and nasal bones may be destroyed by submucous gummata, or by the extension of tertiary ulceration.

THE LONG BONES are rarely attacked by central gummata. In this case spontaneous fracture may occur, which is very slow in uniting.

2. OSTEOSCLEROSIS.—A diffuse osteoperiostitis may affect any of the bones, but especially the tibia, femur, humerus, radius, and ulna. Constant aching pain, worse at night, is complained of, and the subcutaneous parts of the bone become thickened. The X rays show irregular thickening under the periosteum of the whole shaft. The bone remains permanently hard and heavy, and necrosis may take place.

INHERITED SYPHILIS.

THE SKULL presents low nodular elevation over the frontal and parietal eminences—PARROTT'S NODES. These are caused by periostitis forming a layer of spongy new bone. In some places the bones remain partly or wholly unossified—CRANIOTABES. The prominence of the frontal eminences, combined with the depressed nasal bridge, is very characteristic.

The Nasal Septum is destroyed by early suppurative rhinitis, and causes a falling in of the bridge of the nose.

The Palate is narrow, high, and arched, because of the loss of the nasal septum.

Epiphysitis may occur within the first year. An enlargement of the epiphysis takes place, and extends some distance along the shaft. It affects the knees, elbows, or wrists symmetrically, and may lead to separation of the epiphyses or to acute arthritis. Later on it may lead to increased or diminished growth.

Chronic Periostitis, with osteosclerosis, overgrowth, and anterior bowing of the tibia. Hard nodes may form on any of the long bones.

RICKETS.

Etiology.

AGE.—Occurs in first and second years (see also "Late Rickets").

CLIMATE, etc.—Cold, damp, and dark encourage it. Large cities have most of the cases.

DIGESTIVE DISTURBANCES.—Slight diarrhœa and vomiting often precede or accompany rickets.

ERRORS OF DIET are the most constant factors.

In Breast-fed Children it is very rare, but occurs on prolonged lactation, or lactation during pregnancy.

In Hand-fed Children it is common—either when fed on exclusively farinaceous food, or fed with cows' milk in a form in which the casein and fat is vomited.

The Deficiency of Proteid, Animal Fat, and of Earthy Phosphates in the food, is the cause of rickets.

INFLUENCE OF SYPHILIS.—The most severe types of rickets are found in congenital syphilis. Craniotabes, Parrott's nodes, and visceral enlargements are always suspicious of this.

Symptoms—General.

Anæmia, with marked deficiency in red cells.

Muscles are flabby and weak.

Bones are all soft and inclined to bend.

Ligaments soften and stretch.

Tendency to Profuse Sweating, especially over the forehead.

Temperature is subnormal.

Mucous Catarrhs: Gastro-intestinal catarrh—Laryngitis—Bronchitis and bronchopneumonia.

Protuberant abdomen, with large liver and spleen (these are probably cases complicated by congenital syphilis).

Nervous Symptoms: Convulsions—Laryngismus stridulus (laryngeal spasm often precedes a general convulsion)—Tetany. (Painful involuntary muscular contraction. Especially affects the hands and feet. Thumb is drawn into the palm, and fingers form cone-shaped hand).

General Nutrition.—There are two types: one thin, the other fat and flabby.

Rickets, *continued.*

Symptoms—Bones.

CRANIUM.

THIN PATCHES, from deficient inner table = craniotabes.

THICK BOSSES of red vascular bone on parietals, frontals, occipital.

Head is flattened from before backwards.

FOREHEAD is broad, square, and projecting.

FOUR EMINENCES over the frontals and parietals are separated by a + groove.

FONTANELLES (especially the anterior) remain open up to the second or third year.

TEETH erupt late (eleventh or twelfth month). Come in irregular order. Fragile, deficient in enamel, and are shed early. Notched with small segment of a large circle.

THORAX.—Transverse groove at the level of lower end of sternum. Vertical grooves at junction of ribs and cartilages. Prominent sternum. Beading of the ribs where they join the cartilages.

SPINE.—Long curve backwards (kyphosis). Often lateral curve (scoliosis). These often become permanent if they are not corrected before the rickets is cured.

PELVIS.—Contracted and flattened from before backwards. Conjugate diameter, especially at brim, is diminished. Sacro-vertebral angle becomes acuter and more prominent. Anterior superior iliac spines lie as far or farther out than the crests of the ilia.

Triradiate pelvis, caused by pushing in of acetabula, is sometimes found, but is much commoner in osteomalacia.

ARM BONES.—Enlargement of lower radial epiphysis. Radius and ulna bend when child crawls.

LEG BONES.—Enlargement of epiphyses, especially of lower tibial epiphysis. Bending outward and forward of lower third of tibia. Bowing of the femur outwards and forwards. Talipes valgus. Talipes varus rarely, secondary to bowed tibiæ.

Histology.

EPIPHYSEAL CARTILAGE.—Wider in circumference. More vascular than normal. Thicker: about quarter or half an inch as compared with normal one-tenth.

Zone of proliferating cartilage cells is thickened. Cells are irregularly arranged. Cells are increased in proportion to matrix.

Zone of calcified cartilage is thickened. Calcification is very irregular.

Line of ossification is uneven. Islets of cartilage are left in the midst of ossified tissue.

SHAFT.—Subperiosteal layer of bone is more vascular and spongy than normal. Hyperplasia and defective ossification occur on the surface of flat bones. Compact bone and cancellous bone have vessels much larger and more numerous than normal, at the expense of the osseous tissue. Haversian canals are wider than normal.

Result on the Bone as a Whole.—The bone is thicker, but weaker. Contains only 40 per cent earthy matter, as compared with normal 60 per cent. Very liable to greenstick fractures. Bends so that all natural curves are exaggerated.

ON RECOVERY.—The spongy bone becomes somewhat denser than normal. The concavities of the curves are filled by buttresses of new bone, and thus the curves tend to be restored to the normal. Premature ossification of the epiphyseal cartilages leads to stunting of the growth.

Varieties.

FŒTAL RICKETS.—Fractures of the bones, bending of the ribs, hyperplasia of cartilages, are sometimes found when the child is born.

SCURVY-RICKETS.—Often has a sudden onset, with pyrexia. Extreme tenderness of the limbs. Periosteal swellings of hæmorrhagic nature. Especially over the femur and tibia. The epiphyses may be detached, or spontaneous fractures occur. Spongy gums. Ecchymoses and other hæmorrhages, e.g., hæmaturia, epistaxis, or blood-stained diarrhœa, combined with other signs of rickets.

LATE RICKETS.—Occurs about the age of puberty. Is similar to the above, but its effects are confined to the long bones, and the constitutional symptoms are much less marked. Its cause is unknown.

Treatment.

THE DIET should contain fresh proteid and fat in an assimilable form. Fresh milk, properly diluted and peptonized, if necessary, or mixed with lime-water. Raw meat juice, cream and eggs, custard puddings. Also lime-juice, fruit, and vegetables in scurvy rickets.

DRUGS.—These are only of secondary importance. Cod-liver oil in thin children, and phosphate of iron.

SURGICAL.—In the acute stage of rickets deformities should be prevented or corrected by splints, bandages, and manipulations, not allowing the child to walk.

For deformities of the legs left after rickets has ceased, osteotomy will generally be required.

ACHONDROPLASIA.

Definition.—A defective growth of all the bones which develop from cartilage. It differs from rickets in not manifesting its results until some years after birth. It is often hereditary.

Stunting of the Long Bones.—The growth of the shaft of the long bones from the epiphyseal cartilage is very slight, so that the limbs grow in thickness but not in length. The adult is a dwarf.

Achondroplasia, *continued.*

Changes in the Head.—The vault of the skull, which grows from membrane, is normal, whilst the base of the cranium and face are stunted. Mental condition is normal.

Hands.—The stumpy, thick fingers cannot touch one another at their tips, but diverge like the spokes of a wheel.

OSTEOMALACIA.

Definition.—An absorption of the hard parts of the bones, resulting in bending and fractures.

Etiology.—In over 90 per cent it affects women, and usually begins during pregnancy.

Pathology.—The compact parts of the bone are replaced by a fibrocellular tissue, whilst very vascular pulpy marrow fills the medullary cavity. The earthy salts are reduced to a sixth of their normal proportion. Possibly the absorption of the bony salts may be due to the over-production of an internal secretion by the ovaries.

Symptoms.—Severe deep-seated pain in the spine, pelvis, and leg bones. Emaciation and exhaustion. Later the limbs become bent or broken, and the pelvis is pressed into a triradiate shape which makes parturition impossible.

Treatment.—Oophorectomy.

OSTEITIS DEFORMANS.

Definition.—A very rare disease, consisting in late overgrowth of the long bones, with thickening and bending.

Etiology.—Affects middle-aged or old men.

Pathology.—The bones become softened, thickened, and lengthened. The natural curves are exaggerated by bending. The cause is quite unknown. In a few cases multiple sarcomata have grown in the affected bones.

Symptoms and Course.—Severe dull aching pains come and go, and are usually regarded as rheumatic. The onset is very insidious, and the patients live to an old age.

Deformities.

THE CRANIUM becomes thickened and enlarged by an eccentric hypertrophy, the face remaining unaffected.

THE SPINE assumes a long kyphotic curve, with much pain and stiffness.

THE LEG BONES become bowed forwards and outwards.

ACROMEGALY.

Definition.—A bony overgrowth affecting chiefly the jaw, hands, and feet, and leading to gigantism.

Etiology.—Men and women are equally affected, and the disease begins in young adult life.

Pathology.—It is always associated with overgrowth of the anterior half of the pituitary body, which may ultimately lead to death by cerebral compression. The bony changes are merely those of overgrowth.

Symptoms.—Pain is indefinite and intermittent. Headache and lassitude. Some abeyance of the sexual functions, shown by amenorrhœa in women and impotence in men. Appetite and thirst are excessive.

Signs.—The hands and feet enlarge, but more in length than breadth the fingers becoming spatula-shaped. The jaws, especially the lower, become large, and the lips thick. Gigantism is not always present, but nearly all giants are examples of acromegaly.

NEW GROWTHS OF BONES.

Innocent.

FIBROMA (periosteal). Lipoma (periosteal).

CHONDROMA.

OSTEOMA (exostosis).

ANEURYSM by anastomosis. A rare condition, found usually in the crainal bones, producing thinning of the bone over a pulsating tumour.

BLOOD CYSTS. Found in the cancellous ends of the long bones, probably the remains of myeloid sarcomata.

HYDATID CYSTS. May occur in the long bones or the pelvic bones, arising in the medullary cavity without the formation of any parent cyst, the daughter cysts lying free. A chronic thickening of the bone occurs, and later a spontaneous fracture.

Primary Malignant Tumours—Sarcoma.

ENDOSTEAL.—Usually grow from the cancellous tissue at the ends of the long bones. The bone is thinned, and gives the sensation of egg-shell crackling. At first a general enlargement of the bony end occurs, with less pain than in inflammatory disease. Later fracture may occur, and the soft tissues are involved last of all.

1. ROUND- AND SPINDLE-CELLED SARCOMA is very rapid and malignant, diffusing itself along the medulla, and containing masses of cartilage and myxomatous tissue. It quickly causes death by metastatic growths.
2. MYELOID SARCOMA is the commonest form. It occurs in the lower ends of the femur and radius, and the upper ends of the tibia and humerus (i.e., the ends in which the most active growth takes place); also in other epiphyses, the lower jaw, the diplöe of the skull, or as an epulis. It is practically innocent, causing no metastases, and not recurring after local removal. It is extremely vascular, the blood spaces often breaking down to form hæmorrhagic cysts. It often pulsates. A bony skeleton pervades the growth.

Primary Malignant Tumours—Sarcoma, *continued.*

PERIOSTEAL SARCOMA. Is one of the most malignant types of tumour. Round- or spindle-celled. Its growth is rapid and painless at first. It begins on one side of the bone and spreads outside and along it, but at the same time eats into it, and may thus cause a fracture. Some ossification usually takes place in the form of radiating spicules. It is very vascular and pulsating, and large veins form in the skin over it. The edge of the growth is sharply defined and craggy, distinguishing it from the ill-defined swellings of osteitis and periostitis.

TREATMENT OF PRIMARY SARCOMA OF BONES. (1) Amputation high above the disease in all except the myeloid growths. (2) Injection of Coley's fluid. Recurrence or metastases usually take place rapidly.

FOR MYELOID TUMOURS.—Excision of the affected part, or scraping in early cases.

Secondary Malignant Tumours of Bone.

CARCINOMA occurs with the more chronic forms of primary growth, especially with scirrhus of the breast or stomach, also in the skull bones with thyroid cancer. It may cause spontaneous fracture, but with care these fractures re-unite.

SARCOMA is much rarer as a secondary manifestation, because the disease is so rapidly fatal. Spontaneous fractures due to sarcomata never re-unite.

CHAPTER XX.

THE DIAGNOSIS OF SWELLINGS CONNECTED WITH BONES.

Classification.—For diagnostic purposes bone swellings may be grouped as follows.

1. DIATHETIC OR CONSTITUTIONAL.—Rickets—Scurvy rickets—Syphilis—Osteitis deformans—Mollities ossium—Acromegaly.
2. TRAUMATIC.—Fracture—Dislocation—Hæmatoma.
3. SIMPLE OR PYOGENIC INFLAMMATION.—Periostitis—Osteomyelitis—Abscess.
4. SPECIFIC INFECTION.—Syphilis—Tubercle—Actinomycosis—Typhoid.
5. NEW GROWTH.
   SIMPLE.—Exostosis—Enchondroma—Fibroma.
   MALIGNANT.—Sarcoma—Periosteal or central—Carcinoma.
   CYSTS.—Hydatid—Cysts of the jaw—Dermoids.

The Existence of a General Disease.—Rickets, scurvy, scurvy rickets, and congenital syphilis, are all accompanied by manifest and unmistakable symptoms of cachexia.

THE MULTIPLICITY AND DISTRIBUTION OF THE LESIONS indicate the nature of osteitis deformans, mollities, and acromegaly.

The Evidence of Trauma.

ORDINARY FRACTURES AND DISLOCATIONS usually give no difficulty if the cardinal signs are present, and if the measurements, alteration of the relative position of bony points, and skiagram are examined.

IN SPONTANEOUS FRACTURES the trauma may have been very trivial, but in most of these cases the evidence of disunion will be clear. Evidences of tabes or other central nerve disease, or of new growth, should be sought for in obscure cases.

IMPACTED FRACTURES may cause bony swelling with functional disability, but none of the ordinary fracture signs. The locality (head of the humerus, lower end of radius, upper end of the femur), careful measurement, and the skiagram, make the nature clear.

FISSURED FRACTURE with a callus tumour can only be certainly recognized by the skiagram.

The Evidence of Trauma, *continued.*

Hæmatoma connected with bone is rarely seen, except over the cranial bones in the newly-born.

A Long Interval between slight trauma and the appearance of the swelling is suggestive of tuberculous or sarcomatous growths.

The Evidence of Inflammation.—This is clear and unmistakable in all acute pyogenic infections, and indicates periostitis, osteomyelitis, or deep abscess.

The Existence of Severe Constitutional Signs points to infective osteomyelitis or periostitis.

CHRONIC INFLAMMATORY CONDITIONS present the utmost difficulty in distinguishing them from syphilitic, tuberculous, or malignant growths.

The Extensive Thickening of the Shaft of the bone.

The Absence of Any Sharp Edge between the normal and diseased parts.

The Existence of Sinuses leading down to bare bone are the most important characteristic features, but even these do not distinguish chronic pyogenic from syphilitic and tuberculous cases always.

The Evidence of Specific Infection.—This is often hard to obtain in chronic cases, e.g., in diffuse periostitis of syphilis.

In Congenital Syphilis the teeth, palate, nose, and eyes generally give definite evidence of characteristic lesions.

In Acquired Syphilis the history of infection or rash, and the existence of scars on skin or mucous surfaces should be sought for.

In Tuberculous Lesions.—The swelling begins near the epiphysis, and also the tuberculin and ophthalmic reactions are available. (*See page* 52.)

Actinomycosis rarely affects any bones but the jaws, and the granules containing the fungus afford the only proof of its nature.

Typhoid Periostitis.—A clear history of enteric fever.

Simple New Growths rarely give much difficulty.

Exostoses are sharply defined, often pedunculated growths at the epiphyseal junction, or on certain flat bones.

Enchondromata are rare, except in the phalanges of young patients. In other situations they are rarely diagnosed from sarcomata.

Fibroma is a slow, symptomless tumour, growing from one side of the shaft of a bone. It is very rare.

Cysts.

Dermoids are recognized by their locality and by being embedded in a depression in the bone (skull or sternum).

Hydatid Cysts may cause spontaneous fracture, and unless they exist elsewhere are not suspected.

Dental Cysts only affect the jaws.

Malignant Growths.

THE PERIOSTEAL SARCOMATA usually begin on one side of the bone and have a well-defined edge. Their growth is extremely rapid.

THE MEDULLARY SARCOMATA occur commonly in the jaw, upper end of the humerus, lower end of the radius, lower end of the femur, and head of the tibia. Great vascularity, varying consistency at different parts, and egg-shell crackling, are their most characteristic features.

The Region of the Bone affected.

THE DIAPHYSIS is affected chiefly in: Simple and septic inflammation—Syphilitic periostitis—Periosteal sarcoma—Fibroma.

THE REGION OF THE EPIPHYSIS is affected chiefly in: Tubercle—Rickets — Exostoses — Central sarcoma — Abscess (osteomyelitis).

The Physical Characters of the Swellings.

A WELL-DEFINED EDGE indicates a superficial new growth. Great chronicity, with an absence of symptoms or increase, point to this being innocent, whilst rapid growth points to malignancy.

A GRADUALLY SHELVING SWELLING indicates a chronic inflammatory or a central new growth.

EXISTENCE OF SOFT AREAS is suggestive of malignancy.

EGG-SHELL CRACKLING proves the existence of a central expanding growth, generally a myeloid sarcoma, or a cystic tumour of the jaw.

MARKED VASCULARITY.—The superficial veins are large, the skin is hot, and a well-marked bruit exists in many sarcomata.

EXCESSIVE TENDERNESS indicates an inflammatory nature.

SEVERE PAIN, especially at the onset of the disease, with a tendency to nocturnal exacerbation, is present in inflammatory swellings, both simple, pyogenic, and syphilitic. New growths are usually painless in their early stages.

Skiagraphy.

FRACTURES AND DISLOCATIONS are evident. Fissured and impacted fractures are best demonstrated by this method.

CALLUS throws little or no shadow for several weeks after its deposit, i.e., until it has become ossified.

PERIOSTITIS.—In this there is marked thickening of the bone, which may show lamination parallel to the surface. In recent cases it is best seen at the edges of the swelling.

SARCOMATA show spicules radiating at right angles to the bone surface in the less malignant cases. The more malignant conditions show mere bone absorption, with possibly a fracture.

CENTRAL TUBERCULOUS FOCI, GUMMATA, ABSCESS.—Exhibit a light area in the midst of the shaft or epiphysis.

CHAPTER XXI.

INJURIES OF JOINTS.

SPRAINS.*

Definition.—Subcutaneous traumatic injury of soft parts connected with a joint, i.e., of muscles, tendons, and ligaments.

Varieties.

SPRAIN WITHOUT SWELLING.—Otherwise known as a strain. Parts have been stretched and not torn.

SIMPLE UNCOMPLICATED SPRAIN.—Muscles, tendons, or ligaments torn.

SPRAIN COMPLICATED BY A FRACTURE.

SPRAIN COMPLICATED BY A NERVE INJURY.

Symptoms.

PAIN.—Localized—due to direct injury. Referred—due to nerve lesion.

SWELLING.—Immediate—due to hæmorrhage: may occur at a distance from injury, e.g., in ham from sprain in thigh.

Remote—due to inflammatory effusion.

NUMBNESS.—Temporary—due to nerve shock. Lasting more than twelve hours—due to nerve lesion.

Diagnosis.

1. FRACTURE must be determined or excluded by X rays.
2. GROSS NERVE LESION. Distant pain, numbness or paralysis.
3. DISLOCATION. Alteration of relations of bony points.

Treatment.

STRAINS, OR SPRAINS WITHOUT SWELLING.

REST, with firm strapping.

MASSAGE begun within twenty-four hours.

VOLUNTARY MOVEMENTS as early as can be made without pain.

PASSIVE MOVEMENTS deferred till end of one week.

SIMPLE SPRAINS WITH IMMEDIATE SWELLING.

REST, with hot fomentations until increase of swelling ceases. Hot lotio plumbi c. opio.

ELASTIC PRESSURE with crêpe bandage or strapping.

VOLUNTARY MOVEMENTS as soon as can be used without pain.

MASSAGE within twenty-four hours of the time that the swelling ceases to increase.

PASSIVE MOVEMENTS as soon as heat subsides. Well-marked superficial hæmatoma should be incised.

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* Bennett, *Brit. Med. Jour.*, Dec., 1906.

SIMPLE SPRAINS WITH LATE SWELLING.

In Soft Parts wait till heat disappears, and then treat as above.

In Joints without Heat and Tension: Treat as above, with addition of iodides and iodine vasogen locally.

In Joints with Heat and Tension: Aspirate—Calcium chloride to diminish effusion—Rest and hot fomentations. When heat and tension disappear, treat as above.

TREATMENT OF SPRAINS WITH A FRACTURE.—If only fissured, and not complete fracture, treat as above.

If fracture has detached piece of bone, put up in splints. Massage and passive movements as early as possible. Voluntary movements only when bone has set.

TREATMENT OF SPRAINS, ASSOCIATED WITH GROSS NERVE LESIONS.

For painful nerve lesions: rest, and not massage.

For numbness: massage, galvanism, or operation.

Remote Results of Sprains.

PREVENTABLE.—Pain and Stiffness of Joints. The late results of immobilization and want of massage and movement.

Muscular Wasting and Relaxation of Joint. Should be prevented by massage and electricity.

Deformity arises from a fracture which has been overlooked.

UNAVOIDABLE IN CERTAIN CASES. Osteo-arthritis—Local paralysis—Tuberculous disease—Infective arthritis—Myositis ossificans.

MYOSITIS OSSIFICANS.—Arises in torn muscles. Slow development. Not painful. Swelling of affected muscles. Stiffness and difficulty in movement.

Treatment.—Prolonged rest (six months). Mass diminishes, but does not disappear.

Diagnosis.—From sarcoma or periostitis.

X rays show mass in muscle away from bone. Absence of pain or heat.

DISLOCATIONS.

Causes.

1. TRAUMATISM.
2. CONGENITAL CONDITIONS.

Causes (theoretical).—Malposition or injury in utero. Failure of one of the joint surfaces to develop, e.g., rim of the acetabulum, iliac portion of the acetabulum, head of the femur. Fœtal disease of the joint or muscles.

Joints Affected.—Hip—(common); jaw, shoulder, wrist, and patella (rare).

3. PATHOLOGICAL CONDITIONS, either—
   1. Distention of capsule by exudation.
   2. Destruction of either articular surface by disease.
   3. Muscular Traction after the destruction of ligaments.

Any of these may occur in: Acute septic arthritis (especially 1); tubercular disease (especially 2 and 3); osteo-arthritis; *Charcot's disease.*

TRAUMATIC DISLOCATIONS.

Causes.

PREDISPOSING.—Shallow articular cavity. Lax muscles and ligaments. Adult age and male sex.

EXCITING.—Violence—generally indirect. Muscular action in lower jaw, patella, or humerus—rare except after previous dislocations.

Varieties.

COMPLETE.—Articular surfaces are completely separated.
INCOMPLETE (subluxation).—Articular surfaces only partly separated.
COMPOUND.—External wound communicates with joint cavity.
COMPLICATED.—Associated with injury of vessels, nerves, or viscera.

Signs.

LOCAL CONTUSION OF PARTS.—Pain—Swelling—Bruising.

RESTRICTED MOBILITY.

DEFORMITY.—Articular surfaces felt in abnormal position. Altered relation of bony points. Altered axes of bones related to the joint.

Anatomy of Dislocation.

LIGAMENTS are torn, especially the capsular ligament.
MUSCLES torn or stretched.
TENDONS stretched, torn or hitched round dislocated surfaces.
VESSELS and NERVES contused or torn.
ARTICULAR CARTILAGE fissured or torn.
BONY POINTS often fractured.
ARTICULAR CAVITY and surrounding parts are filled with blood.

OF OLD UNREDUCED DISLOCATION.

ARTICULAR CAVITY becomes filled with fibrous tissue.
HEAD OF BONE loses its articular cartilage, becomes buried in dense adhesions or surrounded by new false joint.
MUSCLES become adaptively shortened.
TENDONS acquire new attachments. Muscles, tendons, and ligaments are matted in fibrous tissue.
BONE on which movable bone rests atrophies at point of contact. Is heaped up by periostitis round the point of contact.
If the dislocation is kept at rest for several weeks, FIBROUS UNION takes place.
If the dislocated limb is not kept at rest, a FALSE JOINT is formed.

Treatment of Dislocations.

REDUCTION by :

MANIPULATION, to make the head of the bone retrace its way through the hole in the capsule.
EXTENSION, to overcome muscular tension. Preferably under an anæsthetic. Rarely with the aid of pulleys.

OPERATION.—When the above measures have failed. When fracture which prevents retention exists, e.g., forward dislocation of elbow with fractured olecranon. When fracture which prevents reduction exists, e.g., dislocated shoulder and fractured neck of humerus. When complicated by ruptured vessels or nerves.

RETENTION in position of rest.—Rest for one to three weeks according to the size of the joint. Massage and passive movements at the end of first week. Active movements in addition at the end of first, second, or third week.

DIFFICULTIES IN REDUCTION are caused by: Torn capsule embracing neck of the bone—Tonic contraction of muscles—Interlocking of bony points—Tendons and muscles catching round the neck of displaced bone.

Treatment of Old Unreduced Dislocations.

REDUCTION should be attempted up to the end of the third month. Later than four or six weeks it is unlikely to succeed.

DANGERS: to main vessels and nerves. Fracture of the ends of the bone. Tearing of the soft parts.

MASSAGE AND SYSTEMATIC MOVEMENTS are the best treatment: When some mobility exists—Where there is slight pain—In elderly patients.

OPEN OPERATION is indicated: When no movement is possible without pain—Especially in joints of the upper limb—Especially in young and working men.

ATTEMPT AT REPOSITION by division of tendons and adhesions—generally impossible because articular cavity no longer exists.

EXCISION OF THE JOINT—especially useful in the shoulder or elbow, where mobility is of more importance than stability.

SPECIAL DISLOCATIONS.

Lower Jaw.

DIRECTIONS.—Forwards, generally bilateral.

CAUSES.—Yawning—Gags and dental operations.

SIGNS.—Mouth cannot be shut. In unilateral cases the chin is displaced laterally. A depression occurs in front of the ear.

TREATMENT.—Pressure downwards and backwards on the lower molars by the guarded thumbs.

Clavicle.

STERNAL END.

VARIETIES.

Forward. The bone lies on the manubrium sterni.

Backward. Symptoms occur from pressure on the trachea, œsophagus, and vessels.

Upwards. Very rare.

TREATMENT.—Draw the shoulders back and fix with an 8 bandage.

Special Dislocations—Clavicle, *continued.*

ACROMIAL END.

VARIETIES.—Upwards or downwards.

TREATMENT.—Easy to reduce, difficult to maintain. Bandage over the shoulder and under the flexed elbow. Unreduced dislocation is often of little importance. If it gives rise to pain and stiffness, treat by an open operation.

Shoulder.

PREDISPOSING causes.—Shallowness of glenoid cavity. Laxity of capsule. Freedom of movements.

EXCITING CAUSES.—Falls on outstretched hand or elbow. Violent muscular exertion in cases that have been dislocated before.

MECHANISM.—Head of humerus tears through capsule in front or below. Subsequently displaced forwards or backwards.

VARIETIES and anatomy of each:—

1. SUBCORACOID.—Head lies below coracoid process upon the neck of the scapula. Tendon of subscapularis is torn or stretched over the neck of the humerus. Supraspinatus, infraspinatus, and teres minor are either tightly stretched, producing external rotation, or torn (sometimes with great tuberosity), with internal rotation.
2. SUBCLAVICULAR (rare).—Exaggeration of the subcoracoid variety. Great tuberosity and coracoid processes are fractured or their muscles are torn. Head of bone lies below clavicle.
3. SUBGLENOID (rare).—Head of bone lies on axillary border of scapula below the glenoid cavity. Muscles attached to tuberosities are torn. Axillary vessels compressed.
4. SUBSPINOUS DISLOCATION.—Head rests on the posterior aspect of the neck of the scapula. Subscapularis muscle is torn.
5. SUPRACORACOID (very rare).—Acromion (and possibly coracoid) fractured. Head of the bone tears through upper part of capsule.
6. LUXATIO ERECTA (very rare).—Head of bone lies below glenoid cavity.—Arm is stiffly held above the head. Rotator muscles are intact.

SIGNS OF DISLOCATION.

LOCAL CONTUSION.

RESTRICTED MOBILITY.

FLATTENED OUTER BORDER OF SHOULDER.—Straight edge can touch acromion and external condyle.

HEAD OF BONE is felt in an abnormal position.

ELBOW DISPLACED from the side, and cannot touch the chest wall when the hand is on the opposite shoulder.

VERTICAL MEASUREMENT round the axilla is increased.

ANTERIOR OR POSTERIOR FOLD of the axilla is lowered.

MEASUREMENTS.—From acromion to external condyle is unaltered, but there may be slight shortening, especially in the subclavicular. In the subglenoid variety there is an inch lengthening.

SPECIAL SIGNS of the different varieties:—

SUBCORACOID—Head felt below outer end of the clavicle. Elbow is directed outwards and backwards. Shortening absent or very slight.

SUBCLAVICULAR.—Head felt below middle of the clavicle. Shortening may be present. Elbow is far from the side.

SUBGLENOID.—Head is felt in the axilla. Signs of pressure on vessels and nerves. Marked lengthening of the arm. Flexion of elbow from tension on biceps.

SUBSPINOUS.—Head of the bone felt behind, below the acromion. Elbow displaced forwards. Arm rotated inwards.

TREATMENT OF SHOULDER DISLOCATIONS.

1. MANIPULATION under anæsthetic.

   Kocher's Method: Elbow is held to the side. Hand is brought forwards and outwards, so as to externally rotate the humerus and relax the external rotators. Elbow is adducted to the mid-line—this makes the margins of the gap in the capsule tense. Elbow is raised, so as to slacken upper margin of the rent and keep lower tense. Hand is placed on the opposite shoulder, i.e., arm is rotated inwards, to make the head of the humerus slip into capsule. Elbow lowered.

2. EXTENSION.—Traction on the hand whilst counter-extension is made in the axilla by the heel of the operator.

Elbow Joint.

VARIETIES.—Backwards—Forwards—Lateral.

BACKWARD DISLOCATION (the common variety).—The coronoid process is often broken. The distance from the acromion to the condyles is unaltered. The distance of the olecranon from the condyles is increased. The distance of the condyles from the styloid processes is less. The olecranon is unduly prominent. The arm is held flexed.

FORWARD DISLOCATION. (Rare, except as a complication of fractured olecranon).—The forearm is markedly lengthened. The condyles are unduly prominent. The olecranon is obscured.

LATERAL DISLOCATIONS are very rare and usually incomplete.

TREATMENT.—Flexion of the forearm over the operator's knee placed in the bend of the elbow.

THE ULNA ALONE (very rare).—In backward direction.

THE HEAD OF THE RADIUS.

VARIETIES.—Complete—Forward (the common variety)—Backward or outward (very rare)—Incomplete—Subluxation.

Congenital Dislocation of the Hip, *continued.*

POSITION OF LEG.—Thigh is flexed (by traction of iliopsoas), adducted (by traction of adductors), slightly inverted (by traction of the anterior part of the capsule).

Shortening of one to three inches.

Trochanter is much above Nélaton's line, and may be at the level of iliac crest; it is further from the mid-line than normal.

A gap occurs in the perineum between the thighs.

POSITION OF THE PELVIS.—Flexed on the thigh by traction on iliopsoas; consequent great lordosis. Often tilted laterally in unilateral cases.

MOBILITY.—Free mobility, except in abduction. Limb can be drawn down to its full length by traction in the flexed position, especially in early cases.

PAIN.—Usually absent. Fatigue and aching after walking.

Recurrent synovitis is common.

Passive movements are painless.

IN UNILATERAL CASES.—Waddling and rolling are not so marked. Lameness is much more conspicuous. Scoliosis of lumbar region occurs, with convexity on lame side. There is a difference of one to three inches in the length of the two legs.

SKIAGRAM.—Trochanter is on a level with anterior superior iliac spine. Acetabulum is deficient in its iliac segment. Head of the femur is absent or malformed. The angle between the neck and shaft is more open than normal (coxa valga). A ridge of new bone may jut out from the dorsum ilii above the femur, representing the false acetabulum.

PATHOLOGICAL ANATOMY.

ACETABULUM.—Iliac segment is deficient. Cartilaginous rim is absent. It consists of a triangular shallow depression at union of pubis and ischium.

FALSE ACETABULUM.—In old cases that have walked, a rim of new bone may be present on the dorsum ilii, forming a new acetabulum.

HEAD OF THE FEMUR is stunted or absent. The neck is short.

CAPSULE is lengthened and thickened, as it has to bear the body weight.

LIGAMENTUM TERES is long and band shaped, or absent.

DIAGNOSIS from—

COXA VARA.—Comes on in childhood or puberty after child has walked well. Eversion. Limb cannot be lengthened by traction. Skiagram shows deformity of neck.

TUBERCULOUS HIP.—Late onset of symptoms. Pain is much more marked. Rigidity of the joint. Limb cannot be pulled down. Tuberculin reaction.

TRAUMATIC DISLOCATION.—History of difficult labour. Limb cannot be pulled down. Skiagram shows normal bones.

TREATMENT.

In Early Stages Before Displacement has Occurred:

Prolonged rest in bed for six to twelve months. Traction. Limb is abducted. Pressure is applied to the trochanter.

In Early Cases (up to Six Years) where Deformity Exists:

Lorenz' Bloodless Operation: Limb is pulled down, flexed, abducted, and everted. Adductors are ruptured by kneading. Fixed by plaster bandage, including pelvis and thigh: position of extreme abduction and slight eversion. Retained for twelve weeks. Fixed again with less abduction for six months.

In Older Cases (up to Twelve Years):

Divide adductors, hamstrings, fascia lata, and muscles attached to the trochanter. Open joint behind (Hoffa), in front (Lorenz). Make new acetabulum by gouge. Fix limb for few weeks by plaster.

The Patella.

VARIETIES.—Outwards, inwards, or vertical rotation.

Genu valgum or varum are predisposing causes. Muscular violence or direct violence may cause it.

Accompanied by a rupture of a part of the extension of the quadriceps aponeurosis.

TREAT by flexing the thigh, extending the knee, and pressing into place.

The Knee.

VARIETIES.—Backward, forward, or lateral. Usually are incomplete.

DIAGNOSIS is obvious from the deformity.

The Semilunar Fibro-cartilage of the Knee.

CAUSES.—Violent rotation of the body, standing on one leg, as, e.g., when a violent kick is missed. Any sudden twist of the slightly bent knee.

ANATOMY.

Normal.—Both cartilages are attached to the tibia by (1) Their two extremities; (2) Short vertical ligaments—the coronary ligaments to the edges of the tuberosities. The internal cartilage is semicircular, it is firmly attached to the capsule of the joint and to the internal lateral ligament. The external cartilage is nearly circular; the greater part of its edge is separated from the capsule by the tendon of the popliteus muscle. Hence the internal cartilage is more fixed, the external more mobile.

In Rupture.—The internal cartilage is affected twenty times more often than the external. The reasons for this are:

1. The greater fixity of the internal cartilage.
2. The fact that normal slight rotation at the knee joint takes place round the external condyle as a pivot, the internal tuberosity and cartilage moving, whilst the external are stationary.

The Semilunar Fibro-cartilage of the Knee—Anatomy, *continued.*

The cartilage or its ligaments are torn in one of the following ways:—

1. One or both extremities from the tibia.
2. The edge from the capsule and internal lateral ligament.
3. The coronary ligament from the tibia.
4. The cartilage divided into two pieces, either across or parallel to its long axis.
5. The internal lateral ligament itself is often partly torn.
6. A part of the cartilage may be torn right off to form a loose body.

The torn cartilage becomes displaced, so as to be locked between the joint surfaces or in the inter-condyloid notch.

Synovial effusion with synovitis results.

LATE CHANGES IN THE JOINT.—The torn cartilage and ligaments become converted into fibrocartilaginous tags or loose bodies in the joint. Chronic synovitis is caused by repeated attacks of synovial effusion following a locked joint. The ligaments become relaxed and weakened. The synovial membrane becomes thickened and its free edges converted into fibrous fringes.

SIGNS AND SYMPTOMS.—Violent pain in the knee joint, chiefly on the inner side over the lateral ligament.

The knee is locked in a semi-flexed position.

Synovial effusion with its usual signs appears within a few hours.

Later, and in the absence of proper treatment, recurrent attacks of synovitis often occur in any action of flexion and rotation of the knee.

Tenderness is most marked on pressure over the tibial attachment of the internal lateral ligament.

Very rarely a lump or gap can be felt in the position of the cartilage.

TREATMENT.

REDUCTION by flexion and extension of the joint.

IMMOBILIZATION on a back-splint for six weeks, followed by firm bandaging for three months. This treatment should be adopted in all recent cases occurring for the first time.

OPERATION (for all recurrent cases).—If thorough immobilization has been neglected at first, it is useless later. Incision in a transverse direction over the edge of the affected cartilage. Open the joint. Remove completely all injured or displaced parts of the cartilage. Cut away any tags of hypertrophied synovial membrane. Close without drainage.

RETENTIVE APPARATUS (when operation is refused or undesirable in chronic cases).—The mechanism embraces the joint, and whilst allowing flexion does not permit of any rotation.

DIAGNOSIS.

LOOSE BODY IN THE JOINT.—In this the symptoms and signs are similar, but the pain is not so lasting, locking does not occur, and effusion is not so marked.

THE NIPPING OF SYNOVIAL FRINGES in the joint.—In this the thickened synovial membrane can often be felt. There is some history relating to the onset and cause of chronic synovitis.

The diagnosis between these three conditions can often only be made after the joint is opened.

The Ankle Joint.

FOOT MAY BE DISLOCATED: (1) At the ankle joint, with or without fracture; (2) At the astragalo-calcanean joint.

ASTRAGALUS MAY BE DISLOCATED from between the tibia and the os calcis.

DISLOCATIONS OF THE ANKLE JOINT may be:

Outwards (the commonest) or inwards—always combined with Pott's fracture.

Upwards (the rarest)—generally with Pott's fracture.

Backwards or forwards—generally without fracture.

BACKWARD DISLOCATION.—Both lateral ligaments or both malleoli ruptured. Heel projects backwards. Distance from malleoli to heel is increased. Distance from malleoli to toe is diminished.

FORWARD DISLOCATION.—Structures ruptured as above. Heel is much shortened. Distance from malleoli to heel is diminished. Distance from malleoli to toes is increased.

UPWARD DISLOCATION.—Lateral and interosseous ligaments are torn. Malleoli are widely separated. Shortening of the leg.

TREATMENT.—Reduction and fixation as in Pott's fracture. Massage and passive movements from third day.

The Astragalus.

VARIETIES.

FORWARD.—Generally forwards and outwards. May be forwards and inwards.

BACKWARD.

ROTATION on a sagittal axis, combined with forward or backward displacement.

Generally incomplete—rarely complete.

SIGNS.—Prominence of the astragalus is felt on dorsum of the foot in forward dislocations—Above the heel in backward dislocations.

Distance of malleoli from back of heel and from toes is not altered.

Distance of malleoli from sole may be lessened.

TREATMENT.—Manipulation under anæsthetic with knee flexed.

In complete cases or irreducible incomplete cases, open operation—Replace or remove the astragalus.

Astragalo-Calcanean Joint.

Subastragaloid Dislocations.

VARIETIES.

- BACKWARDS (generally).—Backwards and inwards, or backwards and outwards.
- FORWARDS (very rare).

SIGNS of the ordinary backward dislocations.

- Head of astragalus is prominent on the dorsum. Skin may be burst by the tension.
- Distance from malleoli to heel is increased. Distance from malleoli to toes is decreased.
- Foot is plantar-flexed, and inverted like equino varus, or everted like equino valgus.

TREATMENT.—Reduction under anæsthetic.

- Open operation is often necessary because reduction is impossible.
  - Free the tibialis anticus and posticus tendons from neck of the astragalus. If this fails to reduce, excise the astragalus.

CHAPTER XXII.

DISEASES OF JOINTS.

INFLAMMATION OF JOINTS.

Acute Synovitis.

Causes.—Injury, e.g., Sprain or dislocation—Rheumatism and gout—Syphilis and gonorrhœa—Pyæmia.

Anatomy.—Synovial membrane is hyperæmic. Plasma and cells exude (1) into membrane, (2) into the joint. Membrane is thickened and red. Endothelium may be shed.

Fluid in joint is increased—May be bloodstained.

Lymph is deposited on the articular surfaces.

If the endothelial surface has been destroyed, as it generally is in septic cases, a plastic fibro-cellular exudation takes its place. This subsequently becomes organized into adhesions.

If the inflammation is severe the ligaments become softened and relaxed.

If the cause is simple and not septic, the exudation becomes absorbed, with little or no adhesions, except where cartilages or ligaments have been torn.

Signs.

INFLAMMATION. Heat, redness, swelling, and tenderness.

JOINT IS FIXED by muscular spasm. Position of ease assumed; generally slight flexion. Active and passive movements are equally painful.

SYNOVIAL EFFUSION causes a fluctuating swelling, of characteristic shape. Muscles round joint atrophy (in severe and septic cases). Pain is not felt when the part is at rest. Absence of tenderness on pressing the bony points.

Diagnosis.

IN ARTHRITIS there is pain and swelling of bony ends. Pain is worse in active than passive movements. Grating shows destruction of cartilage.

IN TUBERCULOUS DISEASE.—Original injury absent or trivial—Slow onset—Muscular wasting marked—Inflammation slight or absent—Synovial swelling is pulpy, rather than fluid—May be signs of bone affection as in arthritis—Reaction to tuberculin.

IN SPRAINS.—Violent trauma—Immediate swelling—Marked ecchymosis.

IN HÆMOPHILIA.—History of bleeding and bruising—Rapid swelling after trivial injury—Comparatively painless—May be signs of creaking and grating.

Diagnosis of the Nature of Acute Synovitis.—(See "Acute Arthritis.")

Treatment.

1. Immobilization of joint, when effusion is increasing and inflammation is present.
2. Firm bandaging.
3. Aspiration or leeches if great tension exists.
4. Ice or evaporating lotions in early stages. Hot fomentations to relieve pain after twenty-four hours.
5. Massage and passive movements to be used directly the swelling ceases to increase, and when the inflammation has gone.
6. Active movements as soon as they can be done without pain.
7. If adhesions exist so as to prevent movement, treat by weight extension until inflammation disappears. Move under anæsthetic if no inflammation exists.

Chronic Synovitis.

Causes.—Previous acute attack. Causes of acute synovitis acting in a slight degree. Chronic irritation in joint (ruptured or displaced cartilage, loose cartilage, torn synovial fringes, foreign body). Want of tone in muscles and vessels, e.g., after rising from long confinement to bed.

Anatomy.—Great distention of capsule. Stretching of ligaments. Distention of bursæ communicating with the joint. Thickened synovial membrane, the free edges of which are often produced into hypertrophied villous fringes. The cartilage is often rough and fibrillated.

Signs.—Characteristic swelling. Weakness in the joint. Easily fatigued. Little or no pain.

Diagnosis of chronic synovitis to be made from other conditions producing HYDROPS ARTICULI, viz.:

OSTEO-ARTHRITIS.—Marked grating—Several joints generally affected—Lipping of the cartilage rim.

CHARCOT'S DISEASE.—As in the last, with signs of tabes or syringomyelia.

TUBERCULOUS SYNOVITIS.—Very rare as a hydrops—Absence of adequate cause—Marked muscular wasting—Tuberculous reaction.

SYPHILIS (secondary), and GONORRHŒA—The diagnosis rests on a discovery of the original disease.

Treatment of Chronic Synovitis.

1. Fix the joint, and apply firm pressure, with counter-irritation by blisters.
2. Try systematic massage, with hot-air baths.
3. Aspiration, with pressure and rest.
4. Open the joint. Remove foreign body or synovial fringes. Wash out with sterilized water.

Baker's Cysts.

Definition.—Cysts originating from synovial pouches connected with joints.

Causes.—Chronic synovitis—Bursitis of bursæ connected with joints—Osteo-arthritis—Tuberculous synovitis.

Anatomy.—Synovial sac buried in muscles and fasciæ. Connected to joint by a stalk which has an open communication with the joint, but this may become closed later.

Signs.—Fluctuating swelling in the neighbourhood of a joint. The swelling can in some cases be reduced into the joint. The joint itself usually shows signs of chronic synovitis.

Treatment.—Excision if they cause pain.

INFLAMMATORY DISEASES OF JOINTS.

Acute Arthritis.

All the joint structures are inflamed, i.e., synovial membrane, ligaments, cartilages, and bones.

Causes.

SEPTIC INFECTION: (1) By wounds; (2) By blood-stream (infective arthritis)—*Streptococcus pyogenes*.

EXTENSION from infective osteomyelitis: especially in syphilitic infants. Generally staphylococcal.

SPECIFIC FEVERS.—Measles and scarlet fever (streptococcal infection)—Enteric (*B. typhosus*)—Pneumonia (*Pneumococcus*)—Gonorrhœa (*Gonococcus* or mixed infection)—Rheumatism (doubtful organism)—Pyæmia.

Symptoms.—Synovial distention. Fixation of joint by muscular spasm. Slight flexion of joint.

PAIN.—Acute tenderness. All movement is impossible. Active movement is worse than passive. Starting pains when patient falls asleep.

SIGNS OF ACUTE INFLAMMATION.—Heat, redness, and œdema.

ABSCESS formation in the neighbourhood of joint. Abscesses burst and leave sinuses, leading to dead bone.

SEVERE TOXIC FEVER.

DISLOCATION occurs later when the ligaments have given way.

Terminations.

RECOVERY with a movable joint: only possible in cases treated very early.

DISLOCATION following disorganization.

ANKYLOSIS: fibrous or bony.

DEATH from septicæmia or pyæmia in acute stage, or secondary hæmorrhage; hectic or lardaceous disease later.

Acute Arthritis, *continued.*

Anatomy.

SYNOVIAL MEMBRANE is acutely inflamed.

The exudation from the blood-vessels, instead of forming plastic lymph, is killed by bacterial toxins and forms pus.

The endothelium and then the deeper layers are destroyed by this suppurative inflammation.

The surface of the synovial membrane is eventually replaced by granulation tissue.

LIGAMENTS become softened by inflammatory infiltration. Those which are surrounded by synovial membrane (e.g., ligamentum teres in hip, or crucial ligaments in knee) soon disappear, digested by the proteolytic action of leucocytes and bacteria. Others stretch or are ulcerated through.

CARTILAGE.—Colour changes to dull opaque yellow. It is dissolved away at points of pressure. It is raised in flakes from the bone by inflammatory processes creeping from the edges or through perforations in its surface. Ultimately the cartilage is almost entirely replaced by granulation tissue which springs from the bone beneath, and from the surrounding zone of granulations which have replaced the synovial membrane.

BONE.—The articular surfaces become bare of cartilage. Acute osteitis occurs. The bony tissue is absorbed by osteoclasts. The vessels and cellular elements increase, thus producing osteoporosis and caries. Areas of necrosis occur.

THE PERIOSTEUM is acutely inflamed, but extensive necrosis does not occur, because the bony ends have an independent blood-supply.

In chronic cases which survive the acute stage, stalactiform masses of new bone are produced by the inflamed edge of the periosteum.

THE JOINT CAVITY is first filled with turbid serum ; then with pus, and flakes of necrosed cartilage. Is later lined by granulating surfaces which replace the synovial membrane and cartilage. The granulations coalesce and become organized into dense fibrous adhesions.

Treatment.

1. IN EARLY, MILD, AND DOUBTFUL CASES.

REST, and

BIER'S CONGESTIVE TREATMENT.—A rubber bandage is wound round the limb at some distance above the joint, sufficiently tight to obstruct venous and lymphatic return, without producing coldness or loss of pulse. Kept on for twenty hours out of twenty-four. Limb is elevated during the interval. It makes the whole limb fiery red and very swollen and œdematous. It relieves the pain at once. Produces resolution without suppuration in many cases.

2. IN CASES WHERE SUPPURATION HAS PROBABLY OCCURRED.—Immobilization by splints—Weight extension prevents starting-pains—Open the joint freely—Irrigate with sterile salt solution—Drain.
   Bier's congestive method should be applied after the operation, and greatly hastens recovery.
   Change the dressings frequently, with irrigation.
3. AMPUTATION may be required: In toxæmia threatening life—In secondary hæmorrhage—In chronic suppurating sinuses.
4. EXCISION may be required: For ankylosis in a faulty position.

Special Forms of Acute Arthritis.

Rheumatic.—Generally a synovitis only. Complete recovery without ankylosis is the rule. One joint after another is attacked.

Occasionally arthritis of one or more joints occurs. Ankylosis without suppuration results.

Chronic synovitis is sometimes left. Synovial membrane becomes pulpy. Adhesions produce fibrous ankylosis. Recovery may occur after a long time.

Salicylates are most useful in acute cases.

Acute arthritis resisting salicylates should be opened and washed out.

Gout.—Generally attacks big-toe joint first. Attack begins suddenly in the night. Part is red, œdematous, and very tender. Never affects women. Small rather than the large joints are affected. Leads to changes of osteo-arthritis.

CAUSED by a deposit of biurate of soda in the cartilage, ligaments and fibrous tissue of the joint.

ASSOCIATED with uratic tophi round the joints and in the ears.

TREATED by hot fomentations in the acute stage, and lithia, colchicum, and alkalies in the chronic.

Pyæmic Arthritis.—The joints become rapidly distended with pus. Marked absence of pain.

TREAT by opening, washing, and draining.

Enteric Fever.

1. Simple synovitis, probably toxic rather than microbic.
2. True typhoid arthritis, producing destruction of ligaments and dislocation without suppuration. Tends to spontaneous recovery.
   TREAT by maintaining good position. Opening if the effusion becomes great.
3. Mixed or pure pyogenic infections. Symptoms and treatment as for acute arthritis in general.

Pneumococcal Arthritis.—Occurs as a complication of pneumonia, and less frequently of pneumococcal meningitis or peritonitis. Usually in adults.

Pneumococcal Arthritis, *continued.*

JOINT INCIDENCE.—Upper limb rather more frequently affected than the lower. The knee much more often than any other joint. In two-thirds of the cases only one joint is affected ; in about one-third, two or more suffer at once.

ANATOMY.—About 90 per cent suppurate, and the remainder present a sterile synovitis. Three conditions may thus be found.

1. A serous synovitis—the rarest.
2. A purulent arthritis, with thick flakes of lymph and clear fluid.
3. A purulent arthritis, with thick or thin pus.

The pneumococci are found on the surface of the exudate. The cartilages are eroded and the ligaments destroyed in the acute cases, and if the patient lives, an extra-articular abscess forms. There is little tendency to bone-destruction.

PROGNOSIS.—About 70 per cent of cases die from the toxæmia.

TREATMENT.—Incision and drainage. The functional results are good if the patient lives.

Tuberculous Disease of Joints.

Etiology.

PREDISPOSING CAUSES.—Hereditary disposition (of slight importance). Injury to the joint by trauma or other forms of synovitis. Any condition of general debility.

EXCITING CAUSE.—The entrance of the tubercle bacillus. Sometimes this evidently comes from some other primary focus, but more often not.

AGE.—Patients of any age may be affected. Children and growing adults are the most susceptible, because the young cells of the growing parts of the bone form suitable tissue for the development of the disease. Old people are rather more susceptible than middle-aged, because of their lowered vitality.

POOR PEOPLE are specially liable, because of their bad food, defective hygiene, and the neglect of trivial injuries.

Pathology.—The disease primarily affects either the synovial membrane or the ends of the bone. The cartilages and ligaments are only affected secondarily.

SYNOVIAL CHANGES.

INFILTRATION AND DEGENERATION.—Pulpy swelling caused by œdematous thickening of the membrane, which is studded by early tubercles. Caseation of some of the tuberculous foci, which break into the joint or surrounding structures. Suppuration (in the tuberculous sense). This only occurs in some cases ; in those treated early the disease never goes beyond the pulpy thickening or caseation. When it does occur, a condition like that of a chronic abscess is set up, the synovial membrane being replaced by a fibrous layer lined by granulations exuding tuberculous pus.

Regeneration and Repair.—The caseous foci become cretefied or encapsuled by fibrous tissue. The granulations are converted into fibrous tissue. Opposed granulating surfaces grow together to form fibrous adhesions.

Extension always takes place to some of the other tissues.

- Ligaments are attacked, softened, and destroyed very early, because the synovial membrane is so closely related to them.
- Cartilages are invaded by the thick edge of membrane creeping over them and the disease spreading under their edges.
- Bones are attacked first under the edges of the articular cartilages, and when the latter have been destroyed, directly from the joint disease.
- Bursæ which are connected with the synovial cavity are infected, and in chronic cases may form "Baker's cysts."
- The Cavity of the Joint is first distended with synovial fluid, but a true hydrops articuli of tuberculous origin is rare.
- The Pulpy Membrane may be greatly hypertrophied and form fringes which fill up the folds of the joint, and may cause loose bodies from a separation and fibrous thickening of the free extremities.

BONE CHANGES.

Point of Origin: (1) Beneath the articular cartilage. (2) In the epiphysis next to the growing cartilage. (3) In the diaphysis next to the growing cartilage (this is rare, except in the neck of the femur).

Nature.—Tuberculous invasion of the cancellous tissue. Rarefying osteitis or caries of the bone. Necrosis of small islands of bone between or in carious foci. Osteosclerosis or reparative osteitis round the outer margin of disease tend to limit its spread. The usual tuberculous events of caseation or suppuration accompany its development.

Spread.—Under the edges of the articular cartilage into the joint. Through the articular cartilage into the joint. Through the epiphyseal cartilage into the shaft. Through the compact bone of the neck into the tissues outside the joint.

Marginal Outgrowth of spicules of new bone takes place to a slight degree (see "Periosteal Changes").

CHANGES IN THE CARTILAGES.—These are always secondary to synovial or osseous disease.

Primary Bone Disease beginning under the articular cartilage causes their earliest and most complete destruction.

Attacked from their Deep Surface, by the tuberculous granulations in the bone, the latter fungate through them, or more rarely separate off flakes as loose bodies in the joints.

Attacked from their Superficial Surface, as in synovial disease, they become eroded by the overlying tuberculous granulations from their edges towards their centres, and finally quite replaced by this granulating surface.

Tuberculous Disease of Joints—Pathology, *continued.*

CHANGES IN THE LIGAMENTS.—The ligaments also are only secondarily affected, and especially in synovial disease. They are attacked and replaced by tuberculous tissue. They then quickly become soft and disappear.

Ligaments surrounded by synovial membrane will suffer early and completely, because they are attacked on all sides, e.g., the ligamentum teres of the hip, or the crucial ligaments of the knee.

This destruction of the ligaments allows pathological dislocation to take place by the action of gravity or of muscular tension.

CHANGES IN THE PERIOSTEUM.—This is not as a rule much affected, because it ends at the junction of epiphysis and diaphysis outside the area of disease. It may become inflamed and form new spicules of bone like stalactites round the diseased area. This is more likely to happen if secondary pyogenic infection has taken place.

Changes in the Joint as a Whole.—These will depend upon (1) The structures attacked first; (2) The severity of the process; (3) The efficiency of treatment; (4) The anatomical structure of the joint; (5) The occurrence of pyogenic infection.

There are four chief conditions found in the active stage of tuberculous joint disease:

1. DISTENTION OF THE CAPSULE by pulpy synovial membrane and fluid, the ligaments and cartilages being intact.
2. DISLOCATION from destruction of ligaments, or more rarely of the articular surfaces of the bones, aided by neglect of treatment.
3. DESTRUCTION OF THE BONE ENDS.—The cartilages have disappeared, the diseased bones may be carious or necrotic, forming sequestra in the joint, or they may give rise to TUBERCULOUS ABSCESSES which track outside the joint limits.
4. PYOGENIC INFECTION through opened or burst abscesses.

Ultimate Effect upon the Joint may be one of the following:

1. RECOVERY.—This is only possible when the original disease has been limited to the bone, or when a synovial affection has been arrested or excised in a very early stage.
2. FIXATION BY ADHESIONS.—This results from the destruction of synovial membrane and cartilage, and their being replaced by granulations which have formed fibrous adhesions.
3. BONY ANKYLOSIS.—This results from a more or less complete destruction of the articular cartilages, followed by a union of opposed granulating surfaces of diseased bone. The united granulations become ossified.
4. DISLOCATION.—The diseased tissues recover, but the dislocation remains, and usually the parts become fixed in a mass of fibrous tissue.

Symptoms.—The clinical history can conveniently be divided into three stages, preceded by a latent period; but of course all these stages cannot be recognized in every case.

LATENT PERIOD.—A history is given of some slight injury, since which there has been apparently complete or partial recovery, with a disinclination to use the joint.

EARLY, "FIRST" STAGE, OR STAGE OF INVASION of the disease. Here the disease is limited to either the bone or synovial membrane in which it started, and has not spread to the other structures.

(*a*) IN SYNOVIAL DISEASE. A distention of the joint cavity and formation of a "white swelling." The joint is held in a position which allows of the greatest distention of the capsule, e.g., flexion. The muscles in the neighbourhood are wasted. All movement is painful, passive as well as active. Very rarely a fluctuating hydrops articuli may exist.

(*b*) IN BONE DISEASE. The bone may be swollen; it is tender on pressure. Pushing or jarring the joint surfaces together is painful. There is no fixation in any special position, and passive movements are painless. Active movements, or placing any weight on the limb are painful because all active movements "crowd together" the joint surfaces. A skiagram will show a light area in the bone end.

SECOND STAGE, OR STAGE OF FIXATION.—Here the ligaments and cartilages have been infected from the primary focus, and therefore all the joint tissues are affected. All voluntary movement is abolished by pain. The muscles hold the joint by a tonic contraction, in which the most powerful groups gradually overcome the others, thus producing a fixed deformity. Starting pains occur at night, especially when the cartilage is eroded by the relaxation of this tonic contraction, with consequent jarring of the joint surfaces.

THIRD STAGE, OR STAGE OF DESTRUCTION.—The ligaments have given way, the cartilages are lost, and the bones are carious or necrosed, whilst fixation, wasting, deformity, and pain continue. Further destruction of the joint is manifested by (1) Actual shortening of the bones; (2) Abscess formation; (3) Dislocation.

If the abscesses open upon the surface and become septic, hectic or lardaceous disease may ensue.

CONSTITUTIONAL SYMPTOMS.—These may be entirely absent. Loss of appetite, general malaise, with loss of flesh, are often seen in acute or extensive cases. A hectic temperature is rarely seen unless a pyogenic infection has been added. But it sometimes occurs in children, and then indicates extensive bone disease.

Complications and Sequelæ.—Phthisis—Tuberculous peritonitis—Meningitis—General miliary tuberculosis (all rare except the first)—Septicæmia or lardaceous disease when an abscess has become infected with pyogenic organisms.

Tuberculous Disease of Joints, *continued.*

Prognosis.—This is on the whole good. It is bad when (1) Treatment cannot be efficiently carried out; (2) In children under three and patients over fifty; (3) In the presence of any visceral complication; (4) When septic complications have occurred.

Treatment.

1. CONSTITUTIONAL (see section on "Tuberculosis," p. 51).
2. REST. The diseased joint must be protected from movement and from pressure, which aggravate the disease and its consequences. Bed for a short time and splints for a long time. Complete immobilization must be maintained for some time after all pain and tenderness have disappeared. Three to eighteen months is usual.
3. CORRECTION OF DEFORMITY.—This is to be carried out as early in the disease as possible. It must be done gradually, e.g., by weight extension. It aims at placing the joint in the most useful position if ankylosis results, e.g., flexion of the elbow, extension of the hip or knee.
4. WEIGHT (OR ELASTIC) EXTENSION.—This tends to correct early deformities; to counteract the tonic contraction of the muscles which press the joint surfaces together; to prevent dislocation in the later stages. It is specially useful in disesaes of the spine, hip, and knee.
5. LOCAL TREATMENT.—To be carried out in conjunction with the above:—
   - (*a*) COUNTER-IRRITATION by blisters or cautery is especially useful for the relief of pain.
   - (*b*) PASSIVE HYPERÆMIA. Produced by an elastic band above the joint, applied for one hour daily.
   - (*c*) INJECTIONS OF IODOFORM: 1 oz. of a mixture of iodoform 10 parts, water 20, glycerin ad 100.
   - (*d*) OPENING ABSCESSES under careful aseptic precautions. In most cases, however, the presence of an abcess is an indication for radical operative treatment.
6. TUBERCULIN TREATMENT under the control of opsonic indices (p. 52).
7. EXCISION.
   - (*a*) PARTIAL EXCISION or arthrectomy is an operation of very doubtful utility. If it succeeds in curing, then the disease would probably have been curable without any operation. It usually results in recurrence or ankylosis, with subsequent deformity, requiring a complete excision. It consists in removing diseased synovial membrane with a minimum of ligaments and cartilages, so as to preserve a movable joint.

     It is indicated only in young children (under ten) where there is no bone disease, and where careful non-operative treatment is impracticable.

(*b*) COMPLETE EXCISION, i.e., removal of the bony ends, all cartilages, and most of the ligaments, as well as the synovial membrane.

It is indicated (i) In cases in which general treatment is impossible or has failed. (ii) When there is definite evidence of bone disease which has involved the joint. (iii) When abscesses have formed, especially when these arise from bone disease. (iv) To cure ankylosis in joints, e.g., the shoulder or elbow. (v) To correct faulty ankylosis, e.g., in the knee.

The following conditions are also desirable for success: An age between five and fifty. Good general health, especially the absence of serious visceral disease. Absence of septic sinuses.

8. AMPUTATION.—This is rarely called for.

INDICATIONS. In conditions of very bad general health. In elderly patients, especially when the ankle joint is diseased. When incurable sepsis, with possibly lardaceous disease, has supervened. When excision has failed. When so much bone is diseased that excision would leave a useless limb. When two joints are affected in the same limb.

SYPHILIS OF JOINTS.

Joints are very rarely affected by syphilis. There are three types of disease:—

1. **Chronic Synovitis.**—Usually symmetrical; occurs in the late secondary stage; most often in the knee joints. These become distended by effusion, with little or no pain.

2. **Chronic (Gummatous) Arthritis.**—Gummata may be

(*a*) EXTRA-ARTICULAR.—A chronic swelling outside a joint, which softens and bursts externally.

(*b*) INTRA-ARTICULAR.—The synovial membrane is the seat of a local or diffuse gummatous change. A condition is produced very like tuberculous joints. It differs from the latter by being almost painless, and by producing very little destruction of the ligaments and cartilages.

3. **Osteo-chondro-arthritis.**—May occur in the late acquired or in the congenital disease. It resembles osteo-arthritis. The cartilages become fibrillated and eroded. The synovial membrane is thick and pulpy; the bony ends are thickened by periostitis. It differs from osteo-arthritis by causing little pain, and occurring usually in much younger subjects. The cartilages are "punched-out" rather than worn away, and there is no lipping at their edges.

Treatment.—Mercury and iodides by mouth. Firm pressure over mercury ointment applied to the joint.

Excision is seldom required: only in those cases where actual bony destruction has occurred.

OSTEO-ARTHRITIS.

Definition.—This is a chronic joint lesion which is often degenerative rather than inflammatory in its nature. It represents a collection of widely-differing diseases which have this one point in common, that they produce fibrillation and destruction of the cartilage at one part of the joint, with the heaping up of new cartilage at another.

Etiology. The following are the chief groups of causative conditions.

DEPRESSION OF THE VITALITY.—Old age. Constant cold and damp.

TOXIC INFLUENCES.—Some pre-existing infectious disease, e.g., rheumatism, scarlet fever, tonsillitis, or influenza. Auto-intoxication by some morbid digestive products in, e.g., catarrh or ulceration of the intestines. Infection by a short bacillus (Bannatyne) is demonstrable in many cases.

NERVOUS CAUSES.—Any lesion of the nerves supplying the joint, especially an irritative neuritis. A lesion of the cord affecting the posterior or central columns, e.g., tabes or syringomyelia, or spina bifida. Possibly indefinite conditions of general nervous debility predispose to or produce it occasionally.

TRAUMATISM often acts as the exciting cause when the above-mentioned predisposing causes may have been present. A single severe injury may rupture the cartilage and produce a mono-articular osteo-arthritis at once. Long-continued over-strain may bring about the condition slowly (e.g., in certain joints in labouring men).

Pathological Anatomy.

THE CARTILAGES show the first change. The cells proliferate, the matrix becomes fibrillated. The smooth surface is replaced by a velvety or ragged surface. The articular cartilage becomes worn away along the areas of greatest friction. The edges become heaped up like candle gutterings round the margins of the joint.

THE BONES.—Where the articular cartilage has been removed by attrition, the bone becomes hard and polished. Immediately below this the bone is more open in texture and more fatty than normal, i.e., it is undergoing atrophy. A more or less rapid and extensive absorption of the bone end takes place, the actual joint surface remaining sclerosed. The margins of the articular surfaces form projecting osteophytes by the ossification of the cartilaginous nodules. Locking of the joint may result from the exuberance of these osteophytes.

THE SYNOVIAL MEMBRANE may be either atrophied or hypertrophied.

(*a*) ATROPHY.—More common in old people. Results in an absence of synovial fluid.

(*b*) HYPERTROPHY.—Is the commoner condition. A pulpy *œdematous* condition is followed by the formation of massive fibrous fringes ; these may give rise to pedunculated or loose fibrous or cartilaginous bodies, or the fringes may become filled with fat, forming a "lipoma arborescens." The synovial fluid is increased in amount.

ALL THE JOINT TISSUES are thus affected with atrophy and hypertrophy going on side by side ; the ultimate result will depend on the length of the disease and the predominence of one or other process.

CLINICAL GROUPS.

1. CHRONIC MONARTICULAR FORMS.—Often traumatic in origin, frequent in elderly patients. Atrophy of the joint tissues is more evident than hypertrophy. The large joints are specially affected—the shoulder, hip, or knee.
2. CHRONIC POLYARTICULAR FORMS.—Most common in middle-aged women. It results probably from a chronic toxæmia or nerve disease. The hands and feet are affected first, the large joints and spine later. Atrophy and hypertrophy are evenly balanced.
3. ACUTE POLYARTICULAR FORMS.—Most common in young patients. Probably due to bacterial or toxic absorption. The small joints are chiefly affected. It is often preceded by a definite pyrexia. Hypertrophic thickening of the synovial tissues is most prominent.
4. ACUTE FORMS IN CHILDREN (Still's disease).—The lymph-glands and spleen are enlarged. Some pyrexia accompanies the disease. The cartilage and bones suffer less than the synovial membrane.

Symptoms.

THE ONSET is insidious ; usually marked by pain, which recurs in spasms.

THE ORDER of joint affection varies, but usually the wrist and finger-joints, the ankles and tarsal joints, suffer first ; the hip, knee, and shoulder next ; and the spinal joints last. This especially applies to the more acute forms. In the more chronic atrophic forms, one or both shoulders, knees, hips, or the spinal joints, may alone be affected, the incidence being determined by the patient's occupation.

THE CHANGES IN A SINGLE JOINT.

SWELLING of the synovial tissues, with increased fluid, usually without heat.

THE BONY ENDS are diffusely enlarged.

THE ARTICULAR EDGES become everted and lipped.

GRATING occurs in the joint, and is of two kinds :

1. "Snowball-crunching," from the presence of fibrous fringes.
2. Harsh grating, from the rubbing together of bared bony surfaces.

Osteo-Arthritis—The Changes in a Single Joint, *continued.*

The Shape of the joint is that of a spindle.

The Joint Movements are limited by pain, muscular spasm, and lastly by the locking or osteophytes. The joint-stiffness is worse after rest and better after exercise. The joint is held in a position of semi-flexion.

Pathological dislocations are very rare.

The disease is generally symmetrically bilateral.

CHANGES IN THE LIMB OUTSIDE THE JOINT.—The skin is glossy over the joints. The muscles undergo marked atrophy. Muscular cramp and spasms are frequent. Bursal swellings, or synovial swellings (Baker's cysts), affected in the same way as the joint, may arise in its neighbourhood.

CHARACTERISTIC HAND CHANGES.—In the common poly-articular types: The first phalangeal joint becomes spindle-shaped. The wrist is puffy. The fingers undergo ulnar adduction. Heberden's nodes grow as osteophytes from the bases of the phalanges.

FEET CHANGES.—In the more acute forms the ligaments give way, and a condition of valgus and flat-foot results.

GENERAL SYMPTOMS.—The pulse-rate is increased. The limbs are cold and blue from bad circulation and vasomotor changes. Constant sweating without pyrexia. Patchy pigmentation.

Diagnosis.

IN ARTHRITIS, whether acute or tuberculous, if grating occurs in the joint it is intensely painful. The history is short, and the limb absolutely immobilized. Only in the rare cases of tuberculous hydrops articuli is there any difficulty.

IN GOUT, well-to-do males are affected. It begins in the great toe. Very sudden onset. Tophi, etc.

IN TRAUMATISM, the history is of importance, but often traumatism may be preceded or followed by osteo-arthritis. Crepitus of fractures, adhesions, and tenosynovitis all give a sensation like grating. Immobility at the time and rapid recovery characterize most traumatic lesions.

IN NEW GROWTH of the bone ends, one bone only is affected. The joint is free at first.

IN CHARCOT'S JOINTS.—Conspicuous absence of pain. Generally an exaggerated bony outgrowth and marked nerve symptoms.

IN CHRONIC SYNOVITIS.—" Snow-crunching " crepitation may occur, but there is much more effusion and no enlargement or lipping of the bones.

IN CHRONIC RHEUMATISM, there is no evidence of thickened synovial membrane, or of enlarged or lipped bony ends.

Prognosis.—Is almost hopeless. The disease may be arrested, but *hardly ever* cured. It has little or no tendency to shorten life.

Treatment.

GENERAL.—Avoidance of cold and damp. Dry, bracing climate. Generous diet.

DRUGS.—Guaiacol carbonate (10 gr. t.d.s.), saline aperients, all of which act probably by removing intestinal toxins.

Iodides and salicylates for pain. Arsenic and iron for anæmia.

LOCAL.

IN EARLY (ACUTE) STAGES. Firm pressure over warm wool dressing. Stimulating ointment, e.g., Scott's dressing. Massage and passive movements, but rest from active movements.

IN ADVANCED (CHRONIC) STAGES.—Active movements are permitted. Electric baths, thermal baths, etc.

SURGICAL.—Very rarely required. Excision or osteotomy for a locked or crippled joint, e.g., elbow, hip, knee, or jaw. Most suitable for monarticular forms.

CHARCOT'S DISEASE,

Or Neuropathic Arthritis.

Definition.—An advanced condition of osteo-arthritis resulting from a definite nerve lesion, usually in the spinal cord.

Causes.—Tabes dorsalis (in the great majority of cases)—Syringomyelia—Spina bifida—Hemiplegia, paraplegia, or neuritis. Some actual injury, or the ordinary mechanical strain on the joint, may act as the proximate cause.

Anatomy.—All the changes of osteo-arthritis occur in an exaggerated form.

(*a*) THE ATROPHIC FORM shows marked loss of cartilage and bone, and no osteophytes.

(*b*) THE HYPERTROPHIC FORM shows exuberant overgrowth of cartilage and bone at the margin of the joint. Great increase of synovial fluid, and ultimate disorganization of the joint, are marked features.

Symptoms.

NERVE SYMPTOMS.—Lightning pains, loss of knee-jerks, pupil changes, some anæsthesia or paræsthesia.

JOINT SIGNS.—The knee, hip, shoulder, and ankle are affected in the order of frequency given. Very rarely affects more than one joint.

JOINT SIGNS.

Very rapid and painless onset usually occurs.

A great synovial distention is the first feature.

All the signs of osteo-arthritis, viz., enlargement of the bone ends, lipping of the articular margins, coarse grating, quickly follow.

Charcot's Disease—Symptoms, Joint Signs, *continued.*

Marked absorption of bone, with consequent shortening or deformity.

Massive heaping up of new bone as an outgrowth round the articular margins, in the hypertrophic varieties.

Disorganization or dislocation of the joint from a yielding of the ligaments and destruction of the joint surfaces.

Absence of all pain or tenderness is a conspicuous and characteristic feature.

Treatment.—Protection from injury. Aspiration of excessive effusion. Some kind of mechanical support to prevent disorganization of the joint.

HÆMOPHILIA.

Hæmorrhage into the joints causes two varieties of lesion.

1. In the Early Stages: Chronic synovitis.
2. In the Late Stages as the result of repeated hæmorrhages: osteo-arthritis.

Incidence of the Affection.—The joints are affected in the same order of frequency as in the case of ordinary traumatism. The knees much more frequently, then the ankles, wrists, elbows, shoulders, and hips. A blow or strain which in a normal person produces a sprain, in a hæmophilic causes a hæmarthrus. Subsequently recurrent synovitis may occur without hæmarthrus.

Pathology.—The synovial cavity is distended with blood. The blood remains unabsorbed for a very long time. Some deposit of fibrin takes place on all the articular surfaces. The chronic irritation thus set up produces: in the cartilages—fibrillation, absorption, and nodular thickening at the margins; in the synovial membrane—thickening, with the formation of fibrous fringes; in the joint cavity—a tendency to chronic and recurrent synovitis; in the ligaments—relaxation.

Symptoms.—Acute synovial distention, with heat and pain, are the evidences of the first onset of hæmarthrus. This differs from an ordinary synovitis only in its degree, in proportion to the extent of injury and its slowness to subside.

Chronic and recurrent synovitis, especially in the knee joints, usually follows repeated attacks of hæmarthrus.

The usual signs of osteo-arthritis of the chronic variety finally supervene. Well-marked grating is felt, and the edges of the articular surfaces are lipped.

Treatment.—Of the hæmarthrus—complete and prolonged rest, with firm pressure and cold applications. Of the recurrent synovitis—an elastic bandage constantly applied to the joint. Avoid any surgical interference, e.g., tapping.

LOOSE BODIES IN JOINTS.

Varieties.

FIBRINOUS or "melon-seed bodies" formed in conditions of chronic synovitis, sometimes tuberculous.

HÆMORRHAGIC.—By the organization of blood-clot.

CARTILAGINOUS.—By a chipping off of parts of the articular cartilages or of ecchondroses found in osteo-arthritis. They form mulberry-like masses, from the size of a pea to that of a walnut.

BONE.—Usually invested with cartilage; caused by a quiet necrosis, or separated by traumatism.

SYNOVIAL.—The hypertrophied fringes of chronic synovitis may become detached and form fibrous or cartilaginous loose bodies.

Number.—Usually single, may be multiple, and rarely number several hundreds.

Distribution.—Common in the knee, occasional in the elbow, and very rare in any other joint.

Symptoms.—Sudden attack of acute pain, with momentary locking. Often followed by some synovitis. The loose body can often be detected, generally on the inner side of the patella. Chronic synovitis with relaxation of the ligaments supervenes if it is allowed to remain.

Treatment.—Removal through an open incision, which is much easier if the body can first be transfixed.

Diagnosis.—From displaced semilunar cartilage and nipped synovial fringes (*vide supra*).

ANKYLOSIS OF JOINTS.

False Ankylosis.—The fixation or stiffness produced by extra-articular lesions. These are:

1. CONTRACTION OF THE SKIN and soft parts, e.g., after burns.
2. CONTRACTION OF TENDONS in congenital, paralytic, or traumatic conditions, or after long-continued displacement.
3. THE MATTING TOGETHER of the soft parts by an injury or disease.
4. THE OSSIFICATION OF MUSCLES—myositis ossificans.
5. OUTGROWTHS of the bone, e.g., osteomata, chondromata, or sarcomata.

True Ankylosis.—The fixation or stiffness of a joint produced by intra-articular lesions.

1. FIBROUS ANKYLOSIS.—Formed by fibrous adhesions. The fixation is only partial, but attempts at movement produce pain. The causes are:
    (*a*) SYNOVITIS which has resulted in synovial adhesions, especially tubercle, gonorrhœa, rheumatism.

Fibrous **Ankylosis**, *continued.*

(*b*) Traumatism. This generally produces an extra- as well as an intra-articular ankylosis.

(*c*) Arthritis.—The cartilages have been to some extent replaced by granulation tissue, which has organized into fibrous adhesions. Especially seen in tuberculous conditions.

(*d*) Nerve Lesions, e.g., syringomyelia, or the division of, or neuritis of, a peripheral nerve.

(*e*) Long-continued Abnormal Pressure of contiguous bones, e.g., in scoliosis or talipes.

2. BONY ANKYLOSIS.—Formed by the complete destruction of the articular cartilages and the union of adjacent bony surfaces.

(*a*) Acute Suppurative Arthritis, either septic or gonorrhœal.

(*b*) Chronic Tuberculous Arthritis.

Diagnosis of the nature of ankylosis.

IN FALSE OR EXTRA-ARTICULAR ANKYLOSIS there are signs of extra-articular injury or disease, e.g., swelling in the muscles, tendons, or bones. Movement is generally free and painless, but very limited in extent.

IN FIBROUS ANKYLOSIS: A history of some traumatic, inflammatory, or infective disease. Some movement is possible, but is very painful and accompanied by creaking. There is often swelling and thickening of the synovial tissues.

IN BONY ANKYLOSIS there is absolute rigidity of the joint, and attempts at movement cause no pain.

Treatment.

MASSAGE, PASSIVE AND ACTIVE MOVEMENTS, WITH HOT-AIR BATHS.—All evidence of active disease, e.g., increasing swelling, heat, redness, or tenderness, must be absent. Should always be tried in the early stages, after the subsidence of active exudation or disease.

FORCIBLE MOVEMENT UNDER AN ANÆSTHETIC, followed by massage and passive movements, etc. In the same class of case as the above, after other measures have failed. When passive movements are very painful. It is of the utmost importance that all active inflammatory disease should be at an end. It is dangerous in all recent tuberculous cases.

GRADUAL CORRECTION OF THE DEFORMITY by, e.g., weight extension. Specially useful in tuberculous disease of hip and knee.

EXCISION OF THE JOINT TO PRODUCE MOBILITY.—Most often used in ankylosis of the elbow and shoulder. The bone is freely removed, and passive movements are begun a week later.

EXCISION OR OSTEOTOMY, WITH FIXATION.—Used most often in the hip and knee joints. For conditions of bony ankylosis in a bad position, or fibrous ankylosis, which is painful or weak.

DISEASES OF SPECIAL JOINTS.

SHOULDER JOINT.

Incidence of Diseases.—Inflammatory diseases and tuberculosis are rare. Osteo-arthritis is very common. Ankylosis (usually extra-articular) is very common after traumatism, brachial neuritis, and osteo-arthritis. This is due partly to the facility with which the movements of the scapula replace those of the shoulder, and so prevent active and passive movements.

Synovitis produces a fullness of the deltoid and under the axilla ; best marked along the line of the bicipital groove.

Must be distinguished from subdeltoid bursitis, which does not project into the axilla, and which causes little limitation of movements.

Tuberculosis usually begins in the head of the humerus, and may lead to purely extra-articular disease. May take the form of a chronic caries sicca. Abscesses point along the biceps tendon or under the posterior margin of the deltoid.

Treatment is expectant in most cases.

Operation is indicated when abscesses have formed, or where the skiagram shows extra-articular disease. If excision is done in children (very rare), as little of the epiphysis should be removed as possible.

Ankylosis is very common. When other measures have failed, free excision gives good results.

THE ELBOW JOINT.

Synovitis.—Produces a fluctuating swelling behind on either side of the triceps tendon, together with an obscuration of the head of the radius. The joint is held in a flexed and pronated position.

Tuberculosis.—Is very common. The joint cavity is always involved because of the smallness of the epiphyses. Early swelling and position are the same as in synovitis. All three bone-ends become diseased.

Treatment is usually conservative, especially in children.

Opening and scraping carious foci, with the removal of the synovial membrane, is done in adults and in children in whom abscesses have formed.

A formal excision is reserved for cases where painful or useless ankylosis has occurred.

Ankylosis is common after complicated fractures and after suppurative or tuberculous disease.

If the position is one of flexion it is a matter for the patient's opinion whether anything should be done ; if the position is straight, operation is necessary.

Excision, with free bone removal, gives good results.

WRIST JOINTS.

Gonorrhœal Affection, Osteo-arthritis, and Tubercle are common, but all other diseases are rare.

Synovial Disease and distention produces a swelling, chiefly at the back of the wrist, but an extension to the neighbouring tendon-sheaths soon obscures it. The joint is held flexed.

Tuberculous Disease is very chronic and difficult to cure owing to the number of bones and joints involved. Pulpy swelling is well marked behind and at the sides of the joint, and extends downwards over the metacarpals. Pain is constant and severe in the later stages.

Treatment is conservative and palliative to the utmost degree. Only actual abscesses justify operation. Limited erasions should repeatedly be tried before an extensive excision. In the worst cases amputation is required.

THE SACRO-ILIAC JOINT.

Pyæmic and tuberculous affections are practically the only diseases which affect this joint, and the former presents no special features.

Tuberculous Disease usually affects young adults, and is very rare in children.

THE SIGNS.

Puffy Swelling over the joint behind.

Apparent Lengthening of the Leg. The anterior superior iliac spine is lowered on the affected side.

Abscess forms, and points: over the joint behind, in the gluteal region, through the sacro-sciatic foramen behind the trochanter, in the pelvis, into the rectum, vagina, or perineum.

Pressure over the joint, pressing the two ilia together or forcing them apart, is very painful.

SYMPTOMS.—A general sense of weakness on standing, walking, or straining. A sensation of the pelvis breaking in two. Pain is often referred down the sciatic nerve. The signs and symptoms may be quite latent if only the peripheral parts of the joint are affected.

TREATMENT.—Absolute rest until all active disease is ended.

Opening over the joint posteriorly when abscesses have formed, together with evacuation of the abscesses themselves. Free removal of the diseased bone, removing the part of the ilium posterior to the joint if necessary. Closure without drainage.

A jacket extending from the pelvic brim to the axilla for twelve months subsequent to recovery from the operation.

The operation has usually to be repeated several times before final success.

DISEASES OF THE HIP JOINT.

Simple Synovitis is rare. It gives the same signs as the early stage of tuberculous disease, and under rest with extension quickly passes off. Any tendency to chronicity or recurrence should be regarded as suspicious of tubercle.

Acute Septic Arthritis.—Usually pyæmic, or may follow typhoid fever or one of the exanthems.

Great distention of the joint cavity, with dislocation backwards, is the common tendency.

TREATMENT by weight extension and early evacuation of the abscess.

Sub-acute or Chronic Infective Arthritis.—This results from gonorrhœa or some other infection. It ends usually in ankylosis.

TREATMENT.—Weight extension to produce a straight leg (see under "Ankylosis").

Tuberculous Disease of the Hip.

AGE.—Five to fifteen is the commonest age to begin.

PATHOLOGY.—Disease may commence:

1. IN BONE—the commonest site. (*a*) Under the cartilage of the head. (*b*) In the head near the epiphyseal cartilage. (*c*) In the neck near the epiphyseal cartilage. (*d*) In the acetabulum. (*e*) At the junction of either trochanter (rare, and not strictly hip-joint disease).
2. IN SYNOVIAL MEMBRANE.

Wherever it begins, THE DISEASE USUALLY SPREADS TO: Head of the femur—Acetabulum—Synovial membrane.

IT SPREADS OCCASIONALLY: Through the acetabulum to the pelvis. From the neck to the shaft and great trochanter, without involving the joint. From synovial membrane to psoas bursa, and thence into the pelvis.

LIGAMENTS are invaded by tuberculous granulations from the synovial membrane. The ligamentum teres disappears early. The posterior part of the capsule is perforated. The ilio-femoral, or anterior part of the capsule, usually remains, or is the last ligament to give way.

SYNOVIAL MEMBRANE.—Is infiltrated by tubercles. Thickened to form a pulpy swelling.

BONES.—The head becomes carious and stripped of cartilage, or necroses and forms a sequestrum owing to the obliteration of its blood-supply by:

1. Tuberculous focus in the neck.
2. Synovial disease occluding the blood-vessels in the neck.
3. Destruction of vessels in the ligamentum teres.

ACETABULUM becomes carious. May perforate. May be worn away above and posteriorly by pressure. New bone is formed above and behind the carious acetabulum, thus forming the "travelling acetabulum."

Tuberculous Disease of the Hip—Pathology, *continued.*

- NECK. Carious focus which may spread: (1) Into the joint; (2) Into the shaft and trochanter outside the joint. May form a chronic tuberculous abscess.
- JOINT becomes ankylosed:
  - By fibrous ankylosis due to the union of granulating surfaces, with subsequent fibrosis.
  - By true bony ankylosis due to union of carious bony surfaces, with subsequent ossification.
- Joint becomes dislocated: by destruction of the acetabulum; by destruction of the head or neck; by solution of the ligaments; by contraction of the muscles.

SYMPTOMS.

- FIRST STAGE—Stage of synovitis: Pain in hip or in knee due to common nerve supply. Wasting of the thigh muscles. Fullness of inguinal region. Obliteration of the gluteal fold, with flattened nates.
  - LAMENESS. At first only a slight—almost unconscious—limp.
  - POSITION OF THE LEG.—Leg is flexed to relax vertical limb of Y ligament. Abducted to relax oblique limb of Y ligament. Everted to relax ischiofemoral ligament, and owing to the weight of the limb when patient is lying. Apparently lengthened.
  - POSITION OF THE PELVIS.—Tilted down on diseased side so as to bring abducted thigh parallel to fellow. Hence leg is apparently longer. Lordosis to compensate for flexion of the thigh.
  - OBJECTIVE TENDERNESS.—Pain on extension, adduction, inversion, due to strain on capsular ligaments and pressure of joint surfaces together.
  - Pain on "crowding joint surfaces together" by pressing the heel or the great trochanter.
  - Pain on any passive movement, owing to the rubbing of inflamed synovial surfaces.
  - RIGIDITY.—All movements of the joint limited, especially extension, adduction, and inversion.
- SECOND STAGE.—The stage of destruction of cartilage and ligaments. Pain is increased and nocturnal starting occurs. Rigidity is almost absolute.
  - POSITION OF LEG.
    - Flexion.
    - Adduction. Voluntary movement having ceased, only tonic muscular action is present. The adductors, being much stronger than the abductors, gain this position.
    - Inversion. The posterior part of the capsule has given way, and the short external rotators are infiltrated and weakened.
    - Apparent shortening.

Position of the Pelvis.—Lordosis as above. Tilted up on diseased side so as to bring adducted thigh parallel to its fellow; hence scoliosis and apparent shortening.

Third Stage.—Stage of destruction of bone. Pain as in the second stage. Fever and hectic. Position of the leg as in last in an increased degree, with addition of

Actual Shortening due to (1) Carious destruction of the head; (2) Carious destruction of the acetabulum; (3) Dislocation; (4) Necrosis of the head; (5) Fracture of the neck.

Abscess Formation. Abscess points: Inner side of the great trochanter—Gluteal region—Scarpa's triangle—As a psoas abscess—As intrapelvic abscess bursting into rectum, vagina, or bladder, or ischiorectal fossa—Down the leg towards the knee.

If the abscess bursts and becomes septic, hectic fever and amyloid disease may occur.

DIAGNOSIS.

From Primary Pelvic Abscess.

From Knee-Joint Disease: Knee-joint symptoms.

Caries of the Spine, Psoas Abscess: Angular curvature, pain, and rigidity of spine.

Inflamed Psoas Bursa.

Sciatica: Nerve is tender, and flexion hurts more than extension.

Sacro-iliac Disease: Compression of pelvis is painful—Apparent lengthening of leg—Pain over sacro-iliac joint.

In all the above true hip-joint movements are unimpaired.

Also: From Congenital Dislocation: Absence of pain—Often bilateral—Reducibility.

Osteo-arthritis: Elderly patient—Long history—Grating—Often free movement.

Coxa Vara: Eversion is combined with adduction—Great prominence of the trochanter—Absence of tenderness on pressure—Passive movements painless.

DIAGNOSIS OF THE STRUCTURE CHIEFLY IMPLICATED IN HIP DISEASE.

In Synovial Disease passive movements are almost as painful as active. Movement is much more painful than jarring.

In Disease Chiefly Affecting the Bones, active movements are much more painful than passive, because they crowd together joint surfaces by the muscular contraction. Jarring the joint is more painful than passive movements.

TREATMENT.

Conservative Treatment is the general rule, because it gives good results, and because operative treatment almost always necessitates a loss of joint function.

1. Rest in Bed with weight extension is required until the flexion and some of the abduction or adduction have been cured. Usually a month or less.

Tuberculous Disease of the Hip—Treatment, *continued.*

2. A Single Thomas's Hip Splint applied after the limb has been put into good position. A patten must be worn on the good leg, and then the patient can walk on crutches. Care must be taken that it does not slip round to the outer side of the leg. Usually required for six to twelve months, and must not be discontinued until all pain and tenderness are over, and the X rays show no active disease.
3. A Double Thomas' Splint. For young children who cannot use crutches, or in whom the splint slips round to the outer side of the leg.

Arthrectomy without excision is very seldom employed, because conservative measures give such good results in mild cases, and excision is required in most of the advanced cases.

The joint may be opened behind or in front, and the femur temporarily dislocated. All the synovial membrane is dissected away, and carious foci in the femoral neck or acetabulum scraped.

It may be indicated when prolonged conservative treatment is impossible.

Excision is indicated in the third stage of the disease, i.e. :

When abscesses have formed which, on opening, are found to communicate with the joint.

When marked shortening indicates joint destruction.

Also when prolonged conservative measures have failed to cure.

The Anterior Incision outside the sartorius is the most generally useful, because it divides no important blood-vessels, it affords ready access to the joint, and leaves an anterior scar.

The Posterior Incision is most useful when posterior abscesses have formed, or when the head of the bone lies dislocated backwards.

The Removal of all Diseased Structures, especially sequestra in the head and neck of the bone and the acetabulum, is best done by a large flushing gouge.

After-treatment.—The limb should be put up in marked abduction : (1) In order to cause apposition of the stump of the femur and the acetabulum ; and (2) To compensate for the shortened limb by the subsequent pelvic tilting downwards on the affected side. An adductor tenotomy may be necessary to bring this about. The limb must be kept abducted for six weeks by a plaster or other splint.

Amputation is very rarely called for. It is indicated when the knee as well as the hip is involved, when other operations have repeatedly failed, when intrapelvic disease is extensive, and when septic sinuses defy other treatment, or when the limb is painful and useless.

Osteo-arthritis of the Hip is common in old people. Produces shortening of the leg with raising of the trochanter. Flexion and abduction are limited first, and later all the movements become arrested by the locking of osteophytes.

No special treatment is available.

Ankylosis of the Hip.

CAUSES.—Tuberculosis, pyæmia, gonorrhœa, osteo-arthritis, or traumatism.

POSITION.—If no preventive treatment has been employed, flexion, adduction, and inversion will be marked.

TREATMENT.—Nothing is required if the limb is in good position without much shortening.

Gradual correction by weight extension, for false or fibrous ankylosis in a bad position.

Fixation in an abducted position (*vide* after-treatment of excision of the hip for tuberculous disease) is very valuable in cases with much shortening.

Osteotomy through the neck of the femur or below the trochanters is required in all cases of bony ankylosis in a faulty position.

DISEASES OF THE KNEE JOINT.

Incidence of Disease.—The knee joint is more commonly affected by injury and disease in all its varieties (except perhaps gout) than any other joint.

Simple Synovitis.—The joint is held in a semi-flexed position.

Swelling appears at the sides of the patella, above and below at first, and then it assumes a horse-shoe shape, surrounding the patella, except below, in the position of the patellar ligament.

Fluctuation can be obtained from above to below the patella, pressing on the sides of the swelling.

The patella can be made to tap against the femoral condyles. This is best done when the limb is extended and the quadriceps are relaxed.

Tuberculous Disease begins in the bones in children most commonly, and in the synovial membrane in adults.

Rarely bone disease may produce an extra-articular abscess without affecting the joint.

IN THE FIRST STAGE of disease of the joint cavity, the signs of synovial distention as above given are present; but the hollows of the knee are filled by a doughy substance instead of fluid.

Later, especially in children, a spindle-shaped swelling is formed by the knee, the muscles of the thigh and calf being much wasted.

Tuberculous Disease of Knee Joint, *continued.*

IN THE SECOND STAGE, displacement occurs, which in its complete form is fourfold, i.e., displacement of the tibia backwards and outwards, flexion, and external rotation. This is due to the predominant action of the hamstring muscles, and especially the biceps.

THIRD STAGE.—Actual shortening of either femur or tibia, or of both, from absorption, is produced by bone destruction.

Abscesses form, and usually point at the side of the patella, but may track up the thigh or down the leg.

TREATMENT.—The knee joint probably gives worse results than any other to conservative treatment, i.e., the majority of cases relapse after it, and come to an eventual operation.

CONSERVATIVE TREATMENT.—The limb is, if necessary, straightened by a weight extension, and then fixed in a Thomas's knee splint. Bier's treatment may be well combined with this. Probably six to twelve months will be required, but much shorter periods are usually employed. It is indicated as a first measure in all early cases, especially in children; in cases when the disease is purely osseous or purely synovial.

ARTHRECTOMY, i.e., a removal of all the synovial membrane, and but little of the cartilages, gouging out carious bone foci, with an attempt to leave a movable joint.

This very seldom succeeds, and leaves either a weak, painful, fibrous ankylosis, or requires a more radical excision. It is specially adapted for synovial disease in young children.

EXCISION is indicated: (1) When conservative measures have failed; (2) When the disease has progressed to the second and third stages; (3) In almost all adult cases; (4) When faulty ankylosis has occurred.

Bony ankylosis in a straight position should be aimed at. The lateral and crucial ligaments are freely divided and all the synovial membrane and cartilages removed, with all the diseased bone.

Close apposition by mechanical means is very desirable in order to secure bone union.

AMPUTATION is very rarely called for. Septic sinuses with necrosis at the back of the joint may demand it.

ANKYLOSIS usually results from septic or tuberculous arthritis.

Flexion with backward and outward displacement of the tibia with eversion of the leg is the common position. Hyperextension is occasionally seen.

TREATMENT.—Weight extension, aided by hamstring tenotomies in slight fibrous cases. Excision and fixation in bad fibrous ankylosis. Cuneiform osteotomy and rectification in bony ankylosis.

DISEASES OF THE ANKLE JOINT.

Synovial Distention is rare from simple causes. The characters are similar to the early stages of tuberculous disease.

Tuberculous Disease begins in the astragalus, or less commonly in the synovial membrane or tibia or fibula. The swelling appears under the extensor tendons in front of the joint, and at the sides of the tendo Achillis behind. The foot is held in a position of plantar flexion.

The disease is very liable to spread to the other tarsal joints.

TREATMENT.—Conservative measures should be given a prolonged trial.

Scraping out diseased foci through anterior incisions, with REMOVAL OF THE ASTRAGALUS, is required in relapsing or suppurating cases.

AMPUTATION is indicated when other measures have failed, and especially in older patients.

Ankylosis.—This is very common as the result of injury or talipes. If in faulty position an astragalectomy will often enable it to be rectified.

DISEASES OF THE TARSAL JOINTS.

Incidence.—Gonorrhœa, tubercle, osteo-arthritis, and gout are the commonest affections, together with the changes secondary to various forms of talipes.

Tuberculous Disease.—Bones are often primarily affected, especially the os calcis, astragalus, scaphoid, and cuboid.

The joints between the astragalus and scaphoid, the three cuneiforms, and three inner metatarsals communicate with one another so easily that disease of one generally spreads to the others.

The joints between the os calcis and cuboid, and that between the cuboid and two outer metatarsals, may be affected singly.

Swelling occurs over the affected joints, and is diffuse. Tenderness is marked over affected bones.

TREATMENT.—CONSERVATIVE TREATMENT should be tried for prolonged periods.

LIMITED OPERATIONS are only suitable in rare cases when one bone or joint is alone affected, e.g., the astragalus or os calcis, or calcaneo-cuboid joint.

EXCISION OF THE POSTERIOR TARSAL BONES (Mikulicz' excision), fixing the anterior part of the tarsus in a line with the tibia and fibula, after excision of the adjacent cartilage surfaces, may be tried for extensive bone and synovial disease.

AMPUTATION by Syme's method is usually required in cases where other methods have failed, when septic sinuses have arisen, and when the patient cannot afford long conservative treatment.

CHAPTER XXIII.

THE DIAGNOSIS OF AFFECTIONS OF THE JOINTS.

The History.

1. TRAUMATISM.—The nature, time, and degree of injury should be ascertained. Thus in FRACTURES, DISLOCATIONS, and SPRAINS, the injury is great and the disability immediate. A trivial injury, followed by joint affection after a quiescent interval, is characteristic of TUBERCULOUS DISEASE. An injury of moderate degree, which is often indirect, followed by a swelling after a short interval, indicates SYNOVITIS. A trivial injury followed by immediate painless swelling, is suggestive of HÆMOPHILIA.
2. A PRE-EXISTING DISEASE.—One of the exanthemata—e.g., scarlet fever—influenza, or enteric, may cause a SUPPURATIVE ARTHRITIS. Parturition, or any septic ulcer or wound, may precede PYÆMIC ARTHRITIS. Gonorrhœa or gleet occurs before GONOCOCCAL AFFECTIONS of the joints. HÆMOPHILIA.
3. THE PROGRESS OF THE DISEASE.—The history of previous attacks and intermittence in the symptoms is characteristic of INTERNAL DERANGEMENT. Sudden onset, with rapid amelioration, of TRAUMATIC AFFECTIONS. Slow and steady progress, of TUBERCULOUS DISEASE; and great chronicity, with intermittence of the symptoms, of OSTEO-ARTHRITIS.

The Classification of joint affections for diagnostic purposes.

1. TRAUMATIC.—Arising as the direct result of an injury. Fractures involving the joint—Dislocations—Sprains—Separated epiphyses—Traumatic synovitis and arthritis—Hæmarthrus and hæmophilia.
2. INFLAMMATORY.—Presenting the physical signs of inflammation.
   - (*a*) LOCALIZED TO A SINGLE JOINT.—Synovitis and arthritis, (simple or septic). Acute gout. Acute infantile arthritis, pyæmia, and specific fevers (exceptionally attack a single joint).
   - (*b*) INVOLVING MANY JOINTS.—Pyæmia—Specific fevers—Rheumatism—Gonorrhœa—Acute arthritis of infants—Acute osteo-arthritis.

3. CHRONIC SPECIFIC INFECTIONS.
   (*a*) TUBERCULOUS.—Pain, with the absence of other signs of inflammation, and chronicity, with steady progress, are the main signs of this. Also the reaction to tuberculin and the ophthalmic reaction may be useful in obscure cases.
   (*b*) SYPHILITIC.—These are rare and seldom diagnosed in the absence of other evidence of syphilis. Chronic or subacute synovitis or arthritis, with marked absence of pain, is suggestive.
4. DEGENERATIVE or chronic inflammatory.—Conspicuous deformity of the articular ends of the bones, generally with marked grating and increased synovial fluid, are the characteristics of this class, which includes: Osteo-arthritis (the various chronic forms)—Charcot's disease—Chronic hæmophilia—Chronic gout—Chronic synovitis.

Physical Examination of the affected joint. Comparison should always be made with the sound side.

1. SWELLING.—The shape, position, and character of any swelling determine whether it affects the synovial membrane, the bony ends, or adjacent structures, e.g., bursæ or tendon sheaths.
2. INFLAMMATORY SIGNS are redness, heat, tenderness, pain, and œdema. These indicate various forms of synovitis and arthritis. Marked œdema and acute tenderness indicate suppuration. Swelling and tenderness over the bones, with starting pains at night, occur when the cartilages and bones are diseased, i.e., in arthritis, as distinguished from synovitis.
3. MEASUREMENT OF THE GIRTH.—At the joint it indicates the degree of swelling. Above and below the joint it shows whether there is any muscular wasting, such as occurs so conspicuously in tuberculous and osteo-arthritic limbs. Care should be taken in these measurements to take corresponding points on the two limbs, i.e., at a definite distance from a bony point.
4. RELATIVE POSITION OF BONY POINTS.—Two precautions are necessary: (*a*) To take points which accurately correspond to one another on the two sides; (*b*) To have the two limbs in the same position during the measurement.
   IF THE BONY POINTS ARE SITUATED ON THE SAME BONE, e.g., the olecranon and the styloid process of the ulna, an altered measurement shows a fracture or disease of the bone.
   IF THE BONY POINTS ARE SITUATED ON DIFFERENT BONES, altered measurements may show abnormality of any of the bones or joints intervening between the two points, e.g., the distance from the anterior superior iliac spine to the internal malleolus may be due to injury or disease of the ilium, acetabulum, hip joint, neck or shaft of the femur, knee joint, or tibia.
   IF THE BONY POINTS ARE SITUATED ADJACENT TO A JOINT, altered measurements indicate an affection of the joint, or

Physical Examination—Relative Position of Bony Points, *continued.*

the bony ends concerned in it, e.g., the relation of the iliac spine to the trochanter shows the condition of the hip joint or head and neck of the femur.

These measurements are of especial value in traumatic conditions, e.g., FRACTURES AND DISLOCATIONS, and in TUBERCULOUS JOINT DISEASE.

5. PASSIVE MOVEMENTS.—Care should be taken to exclude movements of neighbouring joints, e.g., in the shoulder joint the scapula should be fixed, and in the hip joint the pelvis.

ABSOLUTE PAINLESS FIXATION indicates bony ankylosis.

PARTIAL FIXATION WITH PAINFUL MOVEMENT indicates synovitis, arthritis, or fibrous ankylosis.

MOVEMENT WHICH IS FREE AND PAINLESS, BUT LIMITED, indicates false or extra-articular ankylosis, and often results from fracture or dislocation.

PAINLESS GRATING indicates chronic osteo-arthritis, Charcot's joint, chronic hæmophilia, or chronic synovitis.

PAINFUL GRATING indicates cartilage destruction and bone disease in arthritis, notably in tuberculous conditions.

THE RANGE OF MOVEMENT NOT ALTERING UNDER ANÆSTHESIA indicates organized ankylosis, as distinguished from the early stages of synovitis and arthritis.

6. ACTIVE MOVEMENTS.—Generally correspond with passive movements.

MARKED LOSS OF ACTIVE AS COMPARED WITH PASSIVE MOVEMENTS suggests a nerve injury producing paralysis.

ACTIVE MOVEMENTS BEING MUCH MORE PAINFUL THAN PASSIVE indicates that the joint surfaces, and especially the bony ends, are involved, because every active movement tends to "crowd together" the joint surfaces. In general terms this distinguishes arthritis and bone disease from mere synovial disease.

PRESSING THE BONY ENDS TOGETHER is productive of pain in the same class of case.

7. EXCLUSION OF NEIGHBOURING STRUCTURES.—These are particularly: (*a*) Bursæ; and (*b*) Tendon sheaths. The locality of the swelling and the limited character of painful movements should enable one to exclude these.

CHAPTER XXIV.

INJURIES AND DISEASES OF THE SPINE.

FRACTURES OF THE SPINE.

Causes.

DIRECT VIOLENCE.—Spine breaks at the point struck.

INDIRECT VIOLENCE.—Spine usually breaks at about the junction between the most movable and the most fixed parts, i.e., between the neck and back.

Varieties.

INCOMPLETE FRACTURES.—The continuity of the column is not destroyed.

Spinous processes, transverse processes, and laminæ may be broken and displaced. They are of importance because late affections of the cord may follow. Occur in the cervical or dorsal regions.

COMPLETE FRACTURES or fracture-dislocation.

Fracture-dislocations.

ANATOMY.—Most common in the cervical and upper dorsal regions. The articular processes are always fractured, except occasionally in the cervical region. The body or intervertebral disc is fractured or torn. The ligaments are extensively lacerated. Fracture, comminution, and impaction of the laminæ and spinous processes may occur. The upper fragment is usually displaced forward.

The cord is either crushed between the laminæ above and the bodies below, or it is cut across or transfixed by bony fragments, or its structural continuity is severed, whilst the bones regain their normal position by natural recoil or treatment.

SIGNS.

DEFORMITY OF THE SPINE.—Usually an angular curvature. There may be a mere irregularity in the line of the spinous processes.

SIGNS OF LOCAL TRAUMA.—Bruising, pain, swelling.

CREPITUS is indistinct, and not to be sought for except with the utmost gentleness.

PARAPLEGIA, ANÆSTHESIA, LOSS OF THE REFLEXES (see below).

VASOMOTOR PARALYSIS, with rise of temperature in the parts below the lesion, up to 110° F., or even higher.

Fracture-dislocations—Signs, *continued.*

TROPHIC LESIONS.

1. Acute bed-sores form over the sacrum or buttocks. Less often over the anterior superior spine or heels. They occur about three days after the injury, or at any later date if myelitis arises. Pressure, and fouling with urine or fæces, act as exciting causes. A large deep slough forms rapidly, and the bone is finally exposed.
2. Of the urinary organs. Sloughing may occur in the bladder, urethra, penis, or scrotum. Septic inflammation ascends to the kidney as a pyelitis and pyelonephritis.

AFFECTION OF THE BLADDER MUSCLES: either true incontinence, retention with overflow, or involuntary reflex micturition.

PARALYSIS OF THE RECTUM or its sphincters.

METEORISM from the abdominal wall paralysis.

PRIAPISM.

EMBARRASSMENT OF RESPIRATION by the paralysis of the abdominal muscles and the intercostals.

TREATMENT.

GENERAL.—To be lifted, upon some supporting sheet, and not picked up by the legs and shoulders. Bed should be firm and upon fracture boards. Special care of the lower extremities lest they get burned by hot-water bottles.

REDUCTION under an anæsthetic: If the lesion is below the cervical region; if the paraplegia is incomplete. Is best performed about twenty-four hours after the accident, when shock has passed off.

LAMINECTOMY.—(1) If the injury to the cord is only partial, indicated by a retention of the deep reflexes and of the sense of heat and pain. (2) Lesions of the cervical spine. (3) Lesions of the cauda equina. (4) When paraplegia arises at an interval after the accident. (5) When the neural arch is driven in.

The operation should be done as soon as possible after the injury to the cord is clear. If once degeneration of the cord has begun, it is useless.

SYMPTOMATIC TREATMENT.—(*a*) Alone: when total transverse lesion has occurred; when the deep reflexes are absent; when myelitis with trophic lesions has set in; when the patient's condition is desperate. (*b*) In conjunction with or following any of the above: Retention apparatus, e.g., sand-bags. Patient should be moved as a whole and not twisted. Skin kept dry and clean with spirit lotions and antiseptic powders. The sheets kept smooth and clean. The urethra disinfected and kept covered by an antiseptic dressing. The urine drawn off by sterile rubber catheter where vesical paralysis exists.

Dislocations of the Spine.

OCCUR only in the cervical region, where the horizontal articular surfaces allow this possibility; most commonly between the fifth and sixth cervical vertebræ.

VARIETIES.—Unilateral and bilateral.

DISPLACEMENT.—The upper part of the spine is displaced forwards, the articular processes locking against the lower.

SYMPTOMS AND SIGNS.—Sudden death in complete dislocations in the upper part. Signs of cord injury in most cases. Neuralgia of the nerves at the seat of injury. Some deformity, either forward displacement or rotation.

TREATMENT.—Reduction under an anæsthetic, or open operation when reduction has failed, with, possibly, removal of part of the lower articular processes.

DISEASES OF THE SPINAL CORD AND NERVES WHICH MAY FOLLOW INJURIES.

Varieties.—(1) Traumatic neurasthenia (railway spine); (2) Concussion of the cord (molecular disintegration); (3) Spinal meningitis; (4) Hæmorrhage into or round the cord; (5) Myelitis; (6) Laceration of the cord; (7) Paraplegia.

1. **Traumatic Neurasthenia,** or railway spine, occurs after a severe accident which has caused both mental and physical shock.

THE SYMPTOMS are usually absent or trivial for the first few days after the accident. Pain in the head and back, especially in the lumbar region. Mental changes: a loss of the power of concentration, undue excitability, etc. Some functional disturbance of micturition. Varying and changing areas of partial anæsthesia or hyperæsthesia.

TREATMENT is constitutional—chiefly rest.

2. **Concussion of the Cord.**—A molecular injury or disintegration without visible lesion.

It produces exactly the same SYMPTOMS as a partial or total laceration at the time, but the symptoms (paraplegia) may rapidly disappear. Priapism is said not to occur in concussion. But more frequently the symptoms are permanent.

3. **Spinal Meningitis** may be simple and plastic, or septic.

Localized pain in the back increased by movement. Neuralgic pain along the course of the spinal nerves. Spastic rigidity, with painful cramps. Hyperæsthesia, with exaggerated reflexes.

4. **Spinal Hæmorrhage.**

(*a*) INTRAMEDULLARY.—Hæmorrhage into the central parts of the cord. Most common in the lower cervical region.

Paraplegia of an incomplete type immediately follows the injury. Senses of pain and temperature are often lost without anæsthesia. No signs of irritation of the spinal nerves. Permanent damage caused by spinal degeneration.

Spinal Hæmorrhage, *continued.*

(*b*) EXTRAMEDULLARY.—Hæmorrhage between the bones and dura, and between the dura and cord.

Paralysis comes on gradually at an interval after the injury. Severe pain with muscular spasms are caused by irritation of the roots of the spinal nerves.

Recovery may be expected, as no permanent cord damage results.

5. **Myelitis** may arise from a septic wound or as a part of the result of any special injury.

The usual symptoms of paraplegia are associated with specially marked trophic disturbances, e.g., acute bed-sores, or sloughing cystitis ; rapid atrophy of muscles supplied by the affected segments.

6. **Laceration of the Cord.**—Paraplegia the most constant and earliest result. Meningitis or myelitis often complicate the paraplegia. Spinal hæmorrhage may accompany or cause the paraplegia.

7. **Paraplegia** resulting from spinal injuries.

RELATION OF THE CORD TO THE VERTEBRAL SPINES :—

Fourth cervical nerve segment (diaphragm) corresponds to second cervical spine.

The cervical enlargement (arm) to 3rd–7th cervical spines.

The lumbar enlargement to 10th–12th dorsal spines.

The lumbar segments (front of the thigh, etc.) to 9th–11th dorsal spines.

The sacral segments (the rest of the leg, etc., rectum and bladder) to 11th dorsal–1st lumbar spines.

Cauda equina to 2nd lumbar–2nd sacral spines.

CAUSES OF TRAUMATIC (SPINAL) PARAPLEGIA.

1. IMMEDIATELY following the injury: Concussion (without manifest injury). Contusion with intramedullary hæmorrhage. Laceration of the cord by crushing of penetrating wounds.
2. ARISING AFTER AN INTERVAL : Extramedullary hæmorrhage (twenty-four to forty-eight hours after, without pyrexia). Inflammatory exudation of meningitis (three days or more after, with pyrexia). Pressure of callus or cicatrices (two weeks or more after).

SIGNS OF A TOTAL TRANSVERSE SPINAL LESION.

1. IN THE AREA SUPPLIED BY THE NERVES, BELOW THE LESION.—Complete motor paralysis, followed by late rigidity and contraction of the muscles. Complete anæsthesia, with loss of senses of pain or temperature. Complete and permanent loss of the deep reflexes. Temporary loss of the superficial reflexes. Vasomotor paralysis, with trophic lesions. Visceral paralysis (bladder, rectum, intestines), according to the site.

2. IN THE AREA SUPPLIED BY THE NERVES AT THE SITE ON THE LESION.—Paralysis, with rapid and flaccid atrophy of the muscles. Zone of hyperæsthesia from nerve-root irritation.

SIGNS OF A PARTIAL LESION.—The central parts of the cord may escape injury. In this case the deep reflexes are retained or soon regained; the senses of pain and of temperature are retained; the anæsthesia may only be partial.

In cases of recovery from concussion or contusion the functions of the cord are regained in the following order: Deep reflexes—Sense of pain and temperature—Tactile sensation—Motor power.

Special Features of lesions occurring at different levels of the spine.

1. LOWER LUMBAR AND SACRAL REGIONS.—The cauda equina is crushed.

Paralysis of all leg muscles except the psoas, iliacus, quadriceps extensor femoris, and the adductors.

Paralysis of all the perineal and penile muscles.

Anæsthesia of the perineal and genital regions, also of the foot and the outer side and back of the leg below the knee.

Bladder is paralyzed. Retention at first with overflow, but true incontinence later from atrophy and relaxation of the sphincters.

Rectum. The sphincter is paralyzed, and incontinence of fæces results.

2. LOWER DORSAL AND UPPER LUMBAR REGIONS.—The lumbar enlargement is injured with the origins of the lumbar and sacral plexuses, and the centres for the bladder and rectum.

Total paralysis and anæsthesia of the legs and perineum.

Bladder and rectum, from an injury to their centres, have their sphincters relaxed from the first, with true incontinence of urine and fæces.

Marked tendency to sloughing of the skin and bladder.

3. MID-DORSAL REGION.—In addition to the paralysis and anæsthesia in the last, paralysis and anæsthesia of the abdominal wall and some of the lower intercostal spaces.

Meteorism results from the abdominal paralysis.

A painful girdle at the waist occurs from hyperæsthesia.

Bladder shows retention with overflow, but later a reflex involuntary micturition may occur.

Rectum is not much affected, but constipation is marked from the abdominal paralysis.

4. UPPER DORSAL REGION.—In addition to the above, paralysis of the intercostal muscles.

Bronchitis with congestive pneumonia is very frequent.

Priapism may occur.

Special Features of Lesions, *continued.*

5. LOWER CERVICAL REGION.—In addition to the last, paralysis and anæsthesia of the arms.

   Priapism is almost constant.

   The pupils are narrowly contracted, forming the so-called "spinal myosis," from a cutting of the sympathetic fibres to the pupil, which descend in the cord and then leave by the lower cervical and upper dorsal nerves.

6. AT OR ABOVE THE THIRD CERVICAL.—Death from asphyxia due to involvement of the phrenic (fourth cervical) and the intercostal nerves.

SPINA BIFIDA.

Definition.—A deficiency of the vertebral neural arches, through which a tumour formed by mal-developed cord or membranes protrudes.

Varieties.

MYELOCELE.—Central canal of the cord opens upon the skin surface in the sacral region (incompatible with life).

SYRINGOMYELOCELE.—Central canal is dilated, and the nerves pass outside the cyst.

MENINGOMYELOCELE.—Dilatation of the membranes form a cyst; the cord is attached to its dorsal wall, and the nerves pass through the cyst.

MENINGOCELE.—A cyst formed of spinal membranes posterior to the cord, which is not affected.

SPINA BIFIDA OCCULTA.—No tumour occurs, except perhaps a lipoma, or a dermoid with long hair.

Signs.

TUMOUR in the median line of the spine. Usually over the lumbo-sacral region, rarely in the cervical or dorsal. The tumour is (except in spina bifida occulta) translucent and fluctuating.

PRESSURE ON THE TUMOUR produces bulging of the anterior fontanelle, and convulsions.

THE EDGES OF THE GAP in the vertebral laminæ can be felt.

THE SKIN COVERING THE TUMOUR is thin, red, often covered with dilated vessels or ulcerated.

Complications.—These are most often found in the syringomyelocele.

OTHER CONGENITAL DEFECTS.—Hydrocephalus—Talipes—Trophic ulcers, perforating ulcer—Arthropathies (Charcot's joint) ankylosis of the small joints of toes—Various forms of paraplegia.

Prognosis.—Usually very bad. It is serious according to the variety, in the order named above. The thickness and nutrition of the overlying skin is another great factor in prognosis.

Treatment.

MERE PROTECTION by a pad if the skin is thick and healthy.

PUNCTURE.

PUNCTURE FOLLOWED BY IODINE INJECTIONS (iodine gr. x, potass. iod. gr. xxx, glycerin ℥j).

OPERATION, with an attempt to replace the cord and nerves and sew the membrane and skin over them.

The patient is placed in an inverted position to lessen the escape of fluid. The prospect of success is very slight, except in pure meningoceles, which are rare.

REMOVAL OF A LIPOMA OR DERMOID in spina bifida occulta when it is causing paraplegia.

SACRO-COCCYGEAL TUMOURS.

Origin.—From non-closure or overgrowth of the neurenteric canal.

Varieties.—Dermoid cyst (in connection with the posterior rectal wall; between the rectum and coccyx; on the skin surface over the coccyx or sacrum). Composite tumours chiefly are myxomatous adenoma (probably this is really a teratoma). Lipoma (probably a degenerate dermoid). Teratoma or partially developed twin. Cystic hygroma. Sarcoma.

Treatment.—Removal by dissection.

If this has to be done in infancy the results are very bad.

TUBERCULOUS DISEASE OF THE SPINE (POTT'S DISEASE).

Etiology.—Children most commonly, but it may occur at any age. A strain or blow may act as a predisposing cause.

Anatomy.—Lower dorsal region is the common site. Cervical region rarely affected, except in children. Lumbar region rarely affected, except in adults. There are three different anatomical types :—

1. PERIOSTEAL.—The disease begins beneath the periosteum of the bodies beneath the anterior common ligament. It spreads beneath the ligament from body to body, and so affects a number of vertebræ. It invades the regions of the intervertebral discs.

   It is specially common in adults.

   It produces no angle, but only a slight kyphotic bend.

2. CENTRAL.—The disease begins in the centrum at the point at which the thin epiphysis joins it. It spreads into the centrum, the adjacent disc, and thence to the next centrum, producing caries and softening. The weight of the column above compresses and crushes the soft carious bodies, and produces an angular deformity. This form thus attacks a few (usually two) vertebræ only.

   It is the common type in children.

Pott's Disease—Anatomy, *continued.*

3. LOCALIZED DISEASE limited to one vertebra.—Usually in the lumbar region. Either under the anterior common ligament, or affecting one of the processes. A definite sequestrum may be formed.

    No deformity and very few symptoms except an abscess result.

After its origin, CASEATION, SUPPURATION, or NECROSIS (rarely), takes place.

THE DISEASE SPREADS by abscess formation (see below), or more rarely to the spinal membranes, producing a local thickening, with pressure on, or disease of, the cord.

CURE TAKES PLACE by a falling together of healthy bones so as to obliterate the diseased focus.

ANKYLOSIS, with obliteration of the affected joints, is the last stage.

Signs and Symptoms.

1. PAIN.
    - (*a*) LOCAL PAIN at the site of the disease. Often inconspicuous, but it may be a constant dull ache.

      Is produced by jarring the spinous processes; by pressure on the head or jerks on the feet; by pressure on the transverse processes; by flexion or rotation of the spine.
    - (*b*) REFERRED PAIN, produced by pressure on nerve roots. Neuralgia of any of the spinal nerves, according to the site of the disease.

      Intercostal neuralgia, or an abdominal girdle pain, are much the most frequent of these signs. In children a stomach-ache may be the only complaint.
2. RIGIDITY.—Muscular rigidity in the early stages of the disease, the muscles being held rigid in order to prevent painful movement. Ankylosis produces absolute and painless rigidity later. The spine cannot be flexed or rotated: this is most marked in the upper regions of the spine. In the cervical region the head is supported by the hands.

    In little children, place the patient on his face and lift the feet; the natural dorsiflexion which would be produced is prevented.
3. DEFORMITY.—Angular displacement at the site of the disease is the rule in "central" disease. The bodies fall together, and the spinous process of the vertebra above (dorsal region), or of the vertebra below (lumbar region) forms an angle projecting backwards.

    A backward curve affecting several or many vertebræ usually results from the periosteal type of disease.

    No displacement at all occurs in very limited or in extensive disease which is quite superficial, and it is rare in cervical disease.

Compensatory curves in a forward direction occur above and below the disease in order to maintain the equilibrium of the body.

In the typical extreme angular deformity of the dorsal region, a hump is produced by it; the head is sunk low between the shoulders; the sternum is bent forwards; the ribs are crowded together, and the heart displaced; lordosis exists in the lumbar region.

4. ABSCESS.—A tuberculous suppuration forms beneath the anterior common ligament, and tracks downwards or laterally.

IN THE CERVICAL REGION: A retropharyngeal abscess (*a*) breaks into the pharynx, or spreads into (*b*) The side of the neck; (*c*) The axilla; (*d*) The mediastinum.

IN THE DORSAL REGION the abscess (*a*) Runs down behind the diaphragm into the psoas sheath; (*b*) Follows the dorsal branch of the intercostal vessels and points outside the erector spinæ; (*c*) Follows the main intercostal vessels and points between the ribs; (*d*) Very rarely tracks upwards into the neck.

IN THE LUMBAR REGION the abscess follows (*a*) The lumbar vessels, and presents outside the erector spinæ or in Petit's triangle; or (*b*) Forms an iliac or psoas abscess.

(For further details of these abscesses, see page 10.)

5. PARAPLEGIA.

CAUSED BY: (*a*) A pressure of tuberculous material upon the cord; (*b*) An abscess pressing upon the dura mater; or (*c*) A tuberculous disease of the cord or its membranes.

Rarely if ever is it the result of a narrowing of the bony canal, which is usually larger rather than smaller than normal.

SPECIAL FEATURES.—A zone of painful anæsthesia corresponding to the nerves at the site of disease. Motor weakness of the legs, the toes being dragged and the limbs feeling heavy and painful. Sensation is unaffected at first. The reflexes are exaggerated. Finally complete paraplegia may result, with anæsthesia, paralysis of the sphincters, and painful involuntary contractions.

RAPID DEATH may occur from a bursting of an abscess through the dura mater producing meningitis.

SUDDEN DEATH may result from disease of the atlas or axis producing dislocation with a crushing of the cord.

Prognosis.

GOOD as regards the prospect of life in uncomplicated cases.

BAD SIGNS and causes of death are: Abscesses which become septic—Implication of the spinal cord and its membranes—Septic sores and cystitis — Tuberculous disease of other organs.

Pott's Disease, *continued.*

Diagnosis.—When pain, rigidity, and deformity co-exist, diagnosis is easy. Three groups of cases present difficulty, viz.:—

1. CASES WHERE DEFORMITY IS ABSENT, pain being the chief symptom.

    LUMBAGO, SCIATICA, NEURALGIA.—In all these marked tenderness exists on pressure over the affected muscles or nerves—No tenderness or pain on pressing on the bones—Rapid relief of pain by salicylates, etc., is often seen.

    SPRAINS OR OTHER INJURIES OF THE SPINE. In these there occur: Relation to a recent injury—Ecchymosis—Symptoms rapidly clear up with rest.

    SPONDYLITIS DEFORMANS.—Affects elderly people—Is of very slow development—A large area of the spine is affected—A general kyphotic curve is produced.

    SPINAL OSTEOMYELITIS.—Symptoms are very acute, with high fever.

    ANEURYSM OF THE AORTA.—The pain is intense, constant, and unrelieved by rest. No tenderness exists on pressure on the bones. Other signs of aneurysm may be present.

    MALIGNANT DISEASE OF THE SPINE.—This is usually secondary to some primary focus in the breast or stomach.

    HYDATID DISEASE OF THE SPINE.—This is practically never diagnosed unless other circumstances, e.g., the history or previous hydatid disease, point to it.

    ABDOMINAL DISEASE, especially chronic appendicitis or renal disease, is simulated by the girdle pain in children. In these there are always an absence of spinal signs and a presence of other visceral symptoms.

    DISEASE OF THE SACRO-ILIAC OR HIP JOINTS.—In these, local pain, stiffness, or deformities of the affected joints are discovered on examination.

2. CASES OF DEFORMITY IN CHILDREN.

    RICKETY KYPHOSIS.—In this the curve is a general one. Rigidity is but slightly marked. There are no specially tender points over the spine.

3. CASES IN WHICH AN ABSCESS OCCURS WITHOUT PAIN OR DEFORMITY.—These have to be distinguished from the following:—

    TUBERCULOUS ABSCESSES OF OTHER ORIGIN, e.g.:

    - HIP DISEASE, or sacro-iliac disease. The signs proper to these diseases will be present.
    - DISEASE OF THE ILIAC BONE.—Skiagraphy may indicate the disease, or some local tenderness may occur over the bone.
    - LYMPH-GLANDS (especially in the neck) forming a cold abscess. Enlarged glands can be felt in the vicinity.
    - EMPYEMA.—History, signs and symptoms of pleural and lung disease.

Pyogenic Abscesses, e.g.:

- Perinephric Abcess.
- Appendicular Abscess.
- Abscess from Disease of the Pelvic Organs.
  - There will almost certainly be signs pointing to the diseased viscus, whilst the spine is mobile and free from tenderness. When these abscesses are opened they smell of the *Bacillus coli.*

In all doubtful cases the following aids to diagnosis should be used:—X-ray photographs in different positions. Opsonic index in conjunction with tuberculin injections.

Treatment.

IMMOBILIZATION.

Rest in Bed until the pain goes. This may be combined with extension apparatus or double Bryant splint (in children).

Fixation Apparatus for six to twelve months. Sayres' plaster or poroplastic jacket for disease of the dorsal spine. A stiff collar for cervical disease, or better, a chest jacket provided with a jury-mast from which the weight of the head is hung. Phelps' box, or a double Thomas's hip splint for little children.

Immobilization should be continued until all pain and tenderness has disappeared, and a supporting apparatus should be worn for at least three months longer.

ABSCESSES must be treated according to general principles (see page 11).

PARAPLEGIA.

Rest in Bed with Extension should be applied directly the signs begin; this will arrest the condition in the majority of cases.

Laminectomy is done for those cases which do not improve after a month or six weeks of rest and extension.

Chapter XXV.

HEAD INJURIES.

Injuries of the Scalp may result in: (*a*) Abscess, or (*b*) Hæmatoma.

1. SUPERFICIAL TO APONEUROSIS.—Small and ill-defined—Trivial.
2. SUB-APONEUROTIC.—Large and ill-defined. Only limited by attachments of occipito-frontalis. If septic, may be fatal.
3. SUB-PERICRANIAL.—Defined by cranial sutures. Commonest form in children. Over parietal bone is usual situation. Hard edge makes it resemble depressed fracture.

TREATMENT.—Open the inflammatory, but leave the blood-swellings alone.

Traumatic Cephal-Hydrocele.—Occurs in children, but is very rare. Fluctuating fluid swelling under the scalp. Communicates with subarachnoid space or lateral ventricle through a fracture in the vault. Pulsates with the heart and respiration.

FRACTURES OF THE BASE OF THE SKULL.

Causes.

INDIRECT VIOLENCE acting on the vertex: By radiation of of fracture from the vertex. By compression of the elastic skull, causing it to burst at the weak points at the base.

DIRECT VIOLENCE.—Penetrating wounds through orbit or nose. Blows transmitted through condyles of jaw. Violence transmitted through the vertebral column.

POSITION OF FRACTURES.—Generally transverse through the fossæ. Often run right across the base. Often run from one middle fossa to the opposite anterior or posterior fossa. Generally traverse the nerve foramina. Petrous bone is often involved.

Varieties.

SIMPLE.—Especially in the posterior fossa.

COMPOUND.—The commonest variety.

In anterior fossa, into the nose or orbit.

In middle fossa, into external or middle ear; into nasopharynx; into sphenoidal sinuses.

In posterior fossa, into nasopharynx.

Brain may be concussed, contused, or lacerated.

COMPLICATED.

Meninges may be lacerated. Meningitis, simple or septic, may follow.

Vessels may be contused, with resulting thrombosis, or lacerated.

- Venous sinuses—subdural hæmorrhage.
- Middle meningeal artery—epidural hæmorrhage.
- Internal carotid artery. Aneurysm or fatal hæmorrhage.

Nerves may be damaged or torn : Loss of sense—Paralysis.

Signs of Fractured Base.

DIRECT SIGNS of fracture. Crepitus is very rare. Only present when comminution exists or when cranium is divided into two parts. Should never be sought for.

USUAL SIGNS are those of the complications.

1. Cerebral Concussion or laceration. Usually prolonged coma.
2. Hæmorrhage.
   - Bleeding from the Nose.—From fracture of ethmoid (anterior fossa) ; basi-sphenoid (middle fossa) ; basi-occipital (posterior fossa). May be swallowed and then vomited.
   - Orbital Hæmorrhage (anterior fossa). Ecchymosis (chiefly of the lower lid). Subconjunctival (below the cornea without posterior limit). Proptosis. Orbital aneurysm :—Internal carotid and cavernous sinuses both injured.
   - From Ear (middle or posterior fossæ). Laceration of small vessels in middle ear, together with rupture of drum. Injury of internal carotid or one of the venous sinuses. Injury of drum, cartilage, or meatus may cause slight bleeding independently of fractured base.
   - Into muscles at the back of the neck (posterior fossæ), shown by ecchymosis.
3. Discharge of Cerebrospinal Fluid.—From the nose, or more commonly the ear. Caused by laceration of dura, generally in the internal meatus. Large quantity (one or more pints).
   - Fluid with the following characters : Sp. gr. 1005—Alkaline—Does not coagulate on boiling—Reduces copper.
4. Escape of Brain Substance from the Ear.—Rare, and only in fatal cases.
5. Nerve Lesions.
   - Optic nerve or tract (rare). Blindness.
   - Third, fourth, sixth nerves common. Ocular paralysis.
   - Seventh and eighth. The commonest of all. Facial paralysis and deafness.
   - Ninth, tenth, eleventh, and twelfth rarely involved.
   - Lesion may be immediate, from rupture, often permanent ; or later from callus involvement, generally recovers.

Fractures of Base of Skull, *continued.*

Prognosis of recovery is that of the complications. Thus in uncomplicated cases it is good. In cerebral lesions, in rupture of large vessels, and in septic complications (meningitis or abscess) it is bad.

Prognosis of Completeness of Recovery is seldom good, because of the sequelæ.

Treatment.—Disinfection and dressing of the external auditory meatus. Shave the head. Apply cold by ice-bags or Leiter's tubes. Keep at absolute physical and mental rest for six weeks. No mental work for three to six months.

FRACTURES OF THE VAULT OF THE SKULL.

Varieties.—Fissured (no symptoms or signs unless compound)—Depressed—Punctured.

Depressed and Punctured Fractures.—Generally comminuted. In children depression may exist without fracture. Outer or inner table may alone be broken.

Inner table is generally much more broken than outer because it is less supported; it is broken by a force of less momentum; the breaking force is radiated over a wider area.

VARIETIES:—

SIMPLE OR COMPOUND.

POND.—No sharp depressed edge.

GUTTER.—With sharp depressed edge.

GUNSHOT.—Wound of entry has inner table most damaged. Wound of exit has outer table most damaged. May be associated with fractured base.

Symptoms.

1. CONCUSSION from the blow.
2. COMPRESSION from pressure of bone or exudate.
3. May be NO CEREBRAL SYMPTOMS at all.
4. If COMPOUND and SEPTIC: Symptoms of septic infection—Necrosis of bones or osteomyelitis, pyæmia—Epidural or subdural abscess—Meningitis—Cerebral abscess—Late cerebral compression—Possibly hernia cerebri.

Treatment.

OPERATE in (1) Punctured; (2) Compound depressed; (3) Simple depressed in adults; (4) Gutter depressed in children.

INTRACRANIAL COMPLICATIONS OF HEAD INJURIES.

Varieties.

CEREBRAL: (1) Concussion; (2) Irritation; (3) Compression; (4) Laceration.

INFLAMMATORY: (1) Subcranial abscess; (2) Meningitis; (3) Cerebral abscess; (4) Sinus thrombosis.

HÆMORRHAGE: (1) Venous sinuses; (2) Extradural vessels; *(3) Cerebral* or intradural vessels.

Concussion.

PATHOLOGY.—Engorgement of viscera. Ecchymosis of brain, but no other macroscopic change.

SYMPTOMS.

UNCONSCIOUSNESS, partial or complete.
PUPILS equal, contracted and reacting. In fatal cases they are equal, dilated, and fixed.
SURFACES pale, cold, clammy.
RESPIRATION slow, shallow, irregular.
TEMPERATURE subnormal.
PULSE weak, rapid, and irregular.
REFLEXES present in all but worst cases.
BLADDER AND RECTUM unaffected except in worst cases.

FOLLOWED BY one of the following conditions :

(1) REACTION: Return of consciousness. Rise of temperature to 101°–102°. Vomiting. Some irritability.
(2) DEATH: Deepening unconsciousness. Temperature rises to 104° or over.
(3) CEREBRAL IRRITATION, INFLAMMATION, or COMPRESSION.

Cerebral Irritation.—Especially in injuries to the FRONTAL and OCCIPITAL regions Due to superficial slight lacerations.

SYMPTOMS.—General flexion—Restlessness—Pulse and respiration normal or slightly irregular—Irritability followed by fatuity.

TREATMENT.—Rest—Cold to the head continuously—Bromides, opium—Nourishing diet.

Compression of the Brain.

CAUSES.—Depressed fractures—New growth of bone—Blood (extradural, subdural, intracerebral)—Œdema—Pus (extradural, subdural, cerebral abscess)—Tumours.

SYMPTOMS.—Unconsciousness, absolute.

RESPIRATIONS slow, laboured, and stertorous. Cheeks puffed out. Soft palate paralyzed. Cheyne-Stokes breathing in bad cases.
PULSE full, slow. Becomes rapid and irregular as death approaches.
TEMPERATURE generally raised. More on the paralyzed side than the other.
PUPILS unequal and fixed. Each first contracts, then dilates ; first the one on side of lesion, then the opposite.
MOTOR PARALYSIS, generally hemiplegic.
BLADDER paralyzed, retention.
RECTUM.—Sphincter paralyzed.
REFLEXES DIFFER on two sides generally. On paralyzed side they are increased.

TREATMENT.—Absolute rest in bed—Remove the cause—Venesection—Lumbar puncture.

Intracranial Complications, *continued.*

Laceration of the Brain.

CAUSES.

1. Blows on the head. Common complications of fractures of the skull. Occurs either (*a*) Beneath point struck; or (*b*) At point opposite, from *contre-coup*.
2. Penetrating Wounds, with compound fracture.

ANATOMY.

Immediate.—Laceration or pulping of nerve substance—Pial hæmorrhage—Ecchymosis in brain substance—Bleeding may be into the ventricles.

Later.—Local œdema—Spreading œdema, with increase of the cerebrospinal fluid—Red softening—Yellow softening, or rarely an arachnoid cyst.

SYMPTOMS.—There are none which are really peculiar to laceration but the following conditions are strongly suggestive of it:—

1. Symptoms of concussion merging into those of compression.
2. Symptoms of concussion, with coma, which does not lessen.
3. Symptoms of concussion, followed by those of irritation.
4. Convulsions affecting regular groups of muscles, leaving convulsed parts paralyzed and spreading in a regular sequence.
5. Temperature rising to 104° F. or over is suggestive of laceration of a severe kind.

Intracranial Inflammation.

VARIETIES.—Subcranial (abscess or pachymeningitis); Meningitis (acute or chronic, diffuse or local); Abscess in the brain substance; Infective Thrombosis of the sinuses.

Subcranial Abscess.—Pus collects between the bone and dura mater.

CAUSES.—Compound septic fracture. Septic scalp wound and osteomyelitis. Contusion followed by auto-infection. Middle-ear disease.

SIGNS AND SYMPTOMS.—Symptoms of suppuration. Compression of the brain. Unhealthy wound with dead bone or œdematous patch on scalp (Pott's puffy tumour). Localized headache and tenderness. Paralysis or spasm if over motor area.

TREATMENT.—Trephine, evacuate, and drain.

Pachymeningitis.—Local thickening of dura. Probably follows contusion or fracture. Fixed headache, with possibly Jacksonian epilepsy.

Acute Diffuse Meningitis.—Infection by pyogenic cocci, including the pneumococcus.

CAUSES.—Septic wounds of the scalp or orbit. Suppuration in nasal cavities or sinuses. Infection from pharynx or fauces. Extension from otitis media.

SYMPTOMS.—Rapid onset of symptoms forty-eight hours after injury. Severe headache. Vomiting of cerebral type. Photophobia. Cerebral irritability, followed by delirium with sharp cry, and later by signs of cerebral compression. Spasm, and twitchings of muscles. Retraction of the head. High temperature, initiated by a rigor.

IN MENINGITIS OF THE CONVEXITY, convulsions affecting special groups of muscles.

IN MENINGITIS OF THE BASE, optic neuritis—Strabismus, from paralysis of motor nerves—Marked retraction of the head.

ANATOMY.—Vascular engorgement of dura and pia mater, cerebral substance, and choroid plexuses. Arachnoid is thick and opaque. Turbid serum or pus occupies the subarachnoid space. Convolutions are flattened. Ventricles are distended.

TREATMENT.—Rest and treatment of the general febrile condition. Cold to the head. Venesection and lumbar puncture (draining subtentorial space).

Chronic Meningitis.

CAUSED BY a local head injury.

CONSISTS OF a thickened patch of membranes. May be adherent to the bone above or to the brain below.

SYMPTOMS.—Headache, constant, fixed, and localized—Local tenderness—Possibly traumatic epilepsy—Mental instability—Alcoholism.

TREATMENT. — Prolonged rest. Absence of excitement. Abstinence from alcohol. Blistering the scalp. Small doses of mercury. Trephine if locality is definite.

Infective Thrombosis of the cerebral sinuses.

CAUSES.—Middle-ear disease (lateral sinus). Septic wounds of scalp and orbit. Septic conditions of nose and pharynx. Erysipelas or cellulitis of face or scalp. Osteomyelitis of cranial bones.

VARIETIES.—Lateral sinus—much the commonest—infected from the ear. Cavernous sinus—next commonest—infected from nose, face, or orbit. Longitudinal sinus (rare). Infected from the scalp, face, or nose.

ANATOMY.—Sinus is plugged by soft, adherent, septic blood-clot. Portions of the infected clot form septic emboli. Emboli cause pulmonary infarctions and abscesses. Inflammation of the bone, meningitis, or abscess are probable complications.

SYMPTOMS AND SIGNS.—Existence of some primary septic focus.

HEADACHE: Sudden and severe. Localized tenderness over the affected sinus. Repeated vomiting.

TEMPERATURE rises to 103°–105°, with a bad rigor; continuous, with remission and REPEATED RIGORS. Dyspnœa and cyanosis are marked during the rigors.

PULSE is full, slow, and compressible, but becomes rapid during the rises of temperature.

Infective Thrombosis—Symptoms and Signs, *continued.*

Local pain and other signs in the lungs may point to the existence of INFARCTIONS.

MENTAL CONDITION, drowsy or delirious.

OPTIC NEURITIS and stiffness of neck muscles point to meningitis.

IN LATERAL SINUS THROMBOSIS: Discharge from the ear ceases. Tender swelling over the jugular vein. Dusky congestion of the side of the face. An abscess may form in the neck.

IN CAVERNOUS SINUS THROMBOSIS: Exophthalmos, congestion of the eye and orbit, ptosis, and ocular paralysis with squint.

IN SUPERIOR LONGITUDINAL SINUS: Turgescence of the scalp and forehead. Tenderness over the sinus. Convulsions of one or both legs.

TREATMENT.

FOR LATERAL SINUS.—Trephine on Reid's base line, three-quarters of an inch behind exterior auditory meatus. Tie internal jugular vein first if diagnosis is certain. Turn out the clot. Perform a radical mastoid operation if necessary. Plug.

FOR LONGITUDINAL SINUS.—Trephine above the torcular.

Abscess of the Brain.

CAUSES.

1. TRAUMATISM, e.g., fracture.
    - Early after injury—superficial, contiguous to bone.
    - Late after injury—deep in white matter of the frontal or parietal lobes.
2. INFECTION FROM NEIGHBOURING SEPTIC FOCI.
    - Middle-ear disease: Generally in cerebellum or temporosphenoidal lobe. Sepsis spreads by direct continuity through bones and membranes. More commonly spreads by vessels and occurs beneath the surface.
    - Disease of frontal, sphenoidal, or ethmoidal sinuses.
    - Thrombosis of venous sinuses.
3. BLOOD INFECTION IN PYÆMIA or exanthemata.
4. TUBERCLE may cause a chronic abscess.

ANATOMY.—General single and of a chronic type. May burst into ventricles or cause œdema of brain.

SYMPTOMS.

HEADACHE: General at first; fixed and localized later; often intermittent.

TEMPERATURE normal, or often subnormal.

VOMITING is frequent.

OPTIC NEURITIS. Anorexia, malaise, and constipation.

COMPRESSION SIGNS: Slow or intermittent pulse. Laboured breathing—Cheyne-Stokes type later. Torpor is succeeded by coma. Pupil on the side of lesion is dilated and fixed.

Focal Signs (rare).

Temporosphenoidal: Paralysis or spasm of the opposite side of face; aphasia if the left side is affected.

Cerebellum: Giddiness and tendency to fall towards the lesion. Nystagmus and deviation of eyes to opposite side. Paralysis of the arm on the same side.

TREATMENT.—Turn down flap of soft parts. Trephine the bone with a 1-in. trephine. Make crucial incision through the dura. Probe for pus. Enlarge opening with sinus forceps. Drain and gently wash out. Pack gauze round the drain.

For temporosphenoidal abscess: three-quarters of an inch above external auditory meatus.

For cerebellar abscess: one and a half inches behind external auditory meatus, and one inch below base line.

For combined operation: one inch behind external auditory meatus, half an inch above base line. Lateral sinus lies in the trephine opening. Enlarge upwards and forwards for temporosphenoidal abscess; downwards and backwards for cerebellar.

Diagnosis of Varieties of Intracranial Inflammation.

PRELIMINARY NOTE.—Two or more inflammatory lesions often co-exist, e.g., thrombosis and abscess, or thrombosis and meningitis, etc.

MENINGITIS.—Sudden onset. Cerebral irritation well marked (delirious cry, photophobia, contracted pupils). Rigidity of neck. High temperature without marked remissions. Early development and rapid course. Optic neuritis if the base is affected.

THROMBOSIS.—Sudden onset. Associated with suppurating focus. Rigors repeated. Temperature high but intermittent. Tenderness in the neck. Pulmonary signs.

SUBCRANIAL ABSCESS.—Local pain, tenderness, and œdematous swelling. Compression symptoms steadily increase. High temperature.

CEREBRAL ABSCESS.—Normal or sub-normal temperature. Headache may be the only symptom. Vomiting and optic neuritis. Late onset and of slow development.

CEREBRAL TUMOUR. — Headache, vomiting, optic neuritis (may be the only symptoms). Symptoms develop slowly, but without remission. Absence of septic focus. Absence of general malaise in the early stages.

INTRACRANIAL HÆMORRHAGE.

Varieties.—(1) Extradural—between the dura and cranium; (2) Intra-meningeal—between the brain and the dura; (3) Cerebral—in the substance of the brain.

Intracranial Hæmorrhage, *continued.*

Extradural or Subcranial Hæmorrhage.

CAUSES.—Ruptured middle meningeal artery in the great majority of cases. Rupture or wounds of the venous sinuses or other meningeal vessels.

MECHANISM.—The middle meningeal artery runs in a deep groove or tunnel in the sphenoid, temporal, and parietal bones, and therefore is often torn in fractures of the base or vertex, or b a blow which separates the dura without fracturing the skull. May be on the side opposite to the blow. Usually the anterior branch which suffers. The blood collects between the dura and the skull, driving the former in upon the brain.

SYMPTOMS.—There are typically three stages :—

1. Concussion from the blow, with signs and symptoms of fractured skull; the blood-pressure is low and hæmorrhage is slight.
2. Recovery from unconsciousness with improving pulse. The blood-pressure rises, and bleeding strips the dura from the bone.
3. Gradual coma from cerebral compression by the blood-clot. This comes on within a few hours or days of the return to consciousness.

Or in bad cases the signs of concussion may merge into those of compression without a conscious interval.

Convulsions and paralysis of the arm and face on the opposite side may occur as the coma sets in.

Congestion of the eye of the same side, with proptosis and ocular paralysis, may result from pressure on the cavernous sinus.

Swelling and ecchymosis of the temporal fossa occur from co-existing injury or from leaking of blood through a fracture.

DIAGNOSIS.—Is only possible when the typical sequence of symptoms occurs.

From INTRAMENINGEAL HÆMORRHAGE by the delayed onset of coma.

From CEREBRAL LACERATION by the absence of marked rise of temperature. But this diagnosis is often impossible.

Also a lumbar puncture withdraws clear fluid containing no blood corpuscles.

TREATMENT.—Trephine and clear out the blood-clot, plugging or tying the meningeal artery. This should not be delayed longer than forty-eight hours after the injury.

PROGNOSIS is bad, because the operation is usually delayed too long, so that the compressed brain cannot recover itself.

Intrameningeal Hæmorrhage.

CAUSES.—Injuries of the meningeal vessels especially those of the pia mater, also of the venous sinuses by punctured wounds or fractures of the skull.

ANATOMY.—The blood is spread out between the brain and the dura, compressing the former. If the case recovers, the blood is *absorbed* and an ARACHNOID CYST left.

SYMPTOMS are those of cerebral compression coming on directly after an injury. Convulsions and paralysis will occur if the motor area of the cortex is affected. Late paralysis or Jacksonian epilepsy may occur from the presence of adhesions or a cyst.

DIAGNOSIS is almost impossible from severe concussion and laceration of the brain.

Spinal fluid from a lumbar puncture contains blood cells.

TREATMENT.—All cases in which coma lasts more than twenty-four hours should be trephined in more places than one if necessary. If the dura is found to be blue and bulging, it is opened, and the blood and clot removed.

ON THE REMOTE RESULTS OF HEAD INJURIES.*

PERCENTAGE.—About half of all cases of fractured skull and concussion show some slight permanent sequelæ. Ten to twenty per cent have a marked after-effect. Nearly 10 per cent develop traumatic insanity.

DISPOSITION to mental after-effects is determined by: (1) Natural temperament; (2) Alcoholic habits; (3) Youth or old age; (4) Efficiency of treatment:—operation, and long rest afterwards, lessening the chances of after-effects.

Remote Symptoms.

1. General Cephalgia.—Constant dull pain over frontal, vertical, or occipital regions.
2. Local Cephalgia.—Headache with local tenderness. May be due either to osteosclerosis (when it is cured by trephining), or meningeal adhesions.
3. Painful Scars.—Caused by the adhesion of the scalp to the cranium.
4. Vertigo.—Is left in 22 per cent of fractured skulls, and is due to cerebral instability.
5. Vomiting is often associated with the vertigo.
6. Mental Changes: (*a*) Melancholia, suicide (1–2 per cent); (*b*) Irritability; (*c*) Loss of "nerve"; (*d*) Inability for mental exertion; (*e*) Disturbed sleep; (*f*) Liability to alcoholism and sunstroke.
7. Motor Aphasia only follows injuries to Broca's centre.
8. Amnesia, or loss of memory, occurs very commonly (35 per cent) in a slight degree. It results from mental instability and failure of power of attention. It may be one of three types: (*a*) Loss of memory of the accident and the following days: this is usual; (*b*) Loss of memory of the life previous to the injury: this is rare; (*c*) Loss of memory for events subsequent to the injury: this is common.
9. Special Nerve Injuries.—Loss of smell, inequality of pupils, accommodative asthenopia, nystagmus, optic atrophy, deafness (12 per cent), increased knee-jerks, slow pulse.

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* Crisp English, *Lancet*, 1904, Vol. I, page 485.

Remote Results of Head Injuries, *continued.*

10. Glycosuria.—Temporary is common. Permanent very rare. More frequently follows injuries of the back of the head.
11. HEMIPLEGIA.—Primary or secondary from scar, cyst formation, or sclerosis.
12. TRAUMATIC EPILEPSY.—Occurs in 7 per cent of fracture cases. The majority are idiopathic or general epilepsy, but one-third are of a Jacksonian type.
    - In Jacksonian or traumatic epilepsy the convulsions precede loss of consciousness, and the latter may be slight or absent. The convulsions usually begin in the same group of muscles, i.e., in those related to the affected cortical centre. From this group the convulsions spread to neighbouring groups until, in bad cases, they may become general. The muscles first convulsed remain paralyzed for some time after each convulsion.
    - PROPHYLAXIS.—If all depressed fractures and punctured cranial wounds were operated on, it would seldom occur.
    - TREATMENT.—Operations are usually very unsatisfactory. For the general type they are useless, and in Jacksonian epilepsy the symptoms soon recur.
        - Cases suitable for operation: (*a*) Presenting a definite injury and scar; (*b*) Localized convulsions; (*c*) History of less than two years; (*d*) Absence of family history of epilepsy or insanity.
        - OPERATION.—After exposing the brain and freeing it from the dura, remove clots, cysts, or massive scars. Place gold-leaf between the brain and the dura.
13. TRAUMATIC INSANITY.—Occurs in about 7 per cent. In the majority the injury merely precipitates a condition to which the patient was disposed. In cases where the injury leads directly to insanity, the lesion is generally over the left pre-frontal or parietal regions.
    - TREATMENT.—In cases where there was no predisposition to insanity, operation gives good results.

CHAPTER XXVI.

DISEASES OF THE CRANIUM, BRAIN, AND MIDDLE EAR.

DISEASES OF THE SCALP.

Simple Tumours.

PAPILLOMA.—Commonly multiple ; may require excision.

FIBROMA.—As a huge pendulous mass forms a pachydermatocele, or else a moderate-sized tumour.

TREAT by excision.

SEBACEOUS CYSTS are very common. Move with the scalp freely over the cranium, and there is no depression in the bone. They may suppurate or fungate, when they form a foul exuberant mass.

TREAT by excision.

DERMOID CYSTS occur near the outer canthus of the eye. They are usually attached to the skull, or buried in a cavity in its surface, or attached to the meninges by a pedicle which goes through the skull.

They should be TREATED by excision, for which the skull may have to be opened.

LIPOMA is lobulated and generally under the aponeurosis, and the scalp moves over it. Usually occurs in the frontal region.

NÆVUS (see below).

Malignant Tumours.

EPITHELIOMA.—Usually begins at or near the ear. It may, however, start as a papilloma or sebaceous cyst. It has the usual characters, including the implication of lymph-glands.

TREATMENT on usual lines.

RODENT ULCER.—Usually starts from the face and spreads upwards.

SARCOMA.—Is rare as a primary scalp tumour.

Pulsating Tumours.

NÆVUS.—Especially over the fontanelle, from which it may derive pulsation.

TRAUMATIC ANEURYSM—ARTERIOVENOUS ANEURYSM—ARTERIAL VARIX (temporal)—CIRSOID ANEURYSM.—Most of these occur more commonly in the scalp than elsewhere (see pages 88 and 94).

Diseases of the Scalp—Pulsating Tumours, *continued.*

TRAUMATIC SWELLINGS OF THE SCALP.—Hæmatoma. Cephalhydrocele (see page 224).

INFLAMMATORY SWELLINGS OF THE SCALP.—Abscess, subpericranial, subaponeurotic or superficial (pages 224 and 228).

DISEASES OF THE CRANIAL BONES.

Simple Tumours.

LEONTIASIS OSSEA.—(See page 248).

OSTEOMA.—Usually compact or ivory. Grows from the outer surfaces of the bones or inside the orbit. It has a broad base, abrupt edge, is of slow, painless growth.

TREATMENT.—Removal by chisel if they cause inconvenience.

Malignant Tumours.

SARCOMA.—May be primary or secondary. Begins as: (1) A periosteal spindle-cell sarcoma; (2) A central myeloid growth which expands the bone; (3) A dural, soft, rapid, round-celled growth, which causes great pain and cerebral compression before breaking through the skull.

All are practically inoperable, except possibly the myeloid growth.

CARCINOMA is always secondary, often to thyroid cancer, in which case the tumours often pulsate.

Pulsating Tumours of the Skull.—Any of the above forms of sarcoma or carcinoma may pulsate.

Aneurysm by anastomosis may occur in the cranial diploe.

Acute Inflammatory Swellings.

PERIOSTITIS or OSTEOMYELITIS may complicate any wound, fracture, or contusion. They will lead to (1) Extracranial abscess; (2) Subcranial abscess; (3) Necrosis of the skull; (4) Pyæmia or septicæmia.

TREATMENT is by free opening.

Chronic Inflammatory Swellings.

SIMPLE PERIOSTITIS.—From carrying weights on the head or after a blow.

SYPHILITIC PERIOSTITIS.—Hard and soft nodes, the latter being the commoner (see Chap. XIX).

TUBERCULOUS DISEASE (see Chap. XIX).

PARROTT'S NODES over the frontal and parietal eminences occur in infants with rickets and congenital syphilis. There are other evidences of these diseases.

General Enlargement of the Skull.

HYDROCEPHALUS.—The cranial cavity is increased, but the bones are much thinned, and in infancy the fontanelles and sutures gape. Usually is congenital or infantile in its origin. Caused by a distention of the lateral ventricles. In extreme cases fluctuation and bony crackling may be felt. The forehead becomes protuberant, and the eyes are pushed forwards.

TREATMENT.—Only to be performed in extreme or advancing cases. Tapping the lateral ventricle and leaving a strand of silk ligatures from the ventricle to the subarachnoid space.

SYPHILIS AND RICKETS also cause a general cranial enlargement which is often associated with hydrocephalus.

OSTEITIS DEFORMANS, ACROMEGALY.—Both cause enlargement of the skull, but diminution of the cranial cavity, owing to the thickening of the bones.

TUMOURS CAUSED BY PROTRUSION OF THE BRAIN OR ITS MEMBRANES THROUGH THE SKULL.

Congenital.

MENINGOCELE.—A protrusion of the meninges containing fluid.

ENCEPHALOCELE.—A protrusion of brain substance through a congenital defect in the skull.

HYDRENCEPHALOCELE.—A combination of the above, or a brain protrusion in which is a cavity communicating with the lateral ventricle.

All these are large swellings which pulsate with all respiratory movements. Those containing brain also pulsate with heart movements. They usually spring from the root of the nose, the occiput, or the region of any of the sutures. Round their broad base can be felt the bony margin of skull defect. They are partly reducible, the reduction being accompanied by convulsions. The skin over them is often thin and nævoid.

TREATMENT is usually impossible. In pure meningoceles excision may be possible.

Acquired.

1. SARCOMA OF THE BRAIN OR MENINGES.
2. HERNIA CEREBRI.—Occurs after (*a*) Ineffectual operations for cerebral tumours; (*b*) Injuries or operations on the skull under septic conditions.

   It is CAUSED by the increased intracranial pressure. The mass consists chiefly of œdematous granulations, but true brain substance may also protrude.

   TREATMENT is chiefly (*a*) Preventive in making free outlet for septic discharges when operating for fracture of the skull; (*b*) Pressure must be used with great care; (*c*) Painting with absolute alcohol or collodion; (*d*) Covering with protective shield; (*e*) Removal is only justifiable when is due to a septic process which has come to an end.

CEREBRAL TUMOURS.

Varieties.

MALIGNANT.

GLIOMA.—A sarcoma consisting chiefly of neuroglia.

SARCOMA.—Both of these are ill-defined and non-encapsuled.

SECONDARY CARCINOMA.

INNOCENT.

PSAMMOMA (from the choroid plexus).

CYSTS.—Arachnoid cysts arise after trauma from an absorption of blood-clot. Simple cysts are of very doubtful origin. Hydatid cysts are rare.

TUBERCULOUS MASSES.—Especially in children.

GUMMATA.

Symptoms.

HEADACHE in a fixed locality subject to great exacerbations and becoming steadily worse. Sometimes tenderness on pressure is felt over the tumour region.

VOMITING.—Constantly repeated without nausea or relation to food. Particularly severe in basic or subtentorial tumours.

OPTIC NEURITIS.—Beginning as a congested and then as a "choked disc," and ending in atrophy. Most constant in basic growths.

GIDDINESS, EPILEPSY, LOSS OF MEMORY, and other mental changes, occur with different frequency according to the regions affected.

MENTAL STUPOR, ending in COMA.

FOCAL SYMPTOMS are only produced when certain areas of the brain are affected (see page 239). When a motor area is affected, localized convulsions followed by temporary local paralysis is the rule. The convulsions and paralysis tend to become more widely spread and more permanent.

Treatment.

MEDICINAL TREATMENT is useless, except in the case of gummata, where large doses of iodides should be given.

OPERATIVE.

PRELIMINARY.—Morphia ($\frac{1}{4}$ gr.) is administered beforehand to diminish the shock. The patient has the head and trunk raised. Hæmostasis may be helped by one of two preliminary measures :—

1. Elastic band round the cranium, just over the orbits and ears. This of course only controls the external vessels, and is hardly worth adopting.
2. Temporary clamping of the common carotid artery on the side of the operation.

SKULL OPENING.—May be made in one of two ways :—

1. By first turning down a flap containing all the soft parts, and then removing the bone by trephine, gouge, and forceps. Suitable for small openings and those in the temporal or cervical regions.

2. Turning down an osteoplastic flap containing scalp and cranium, cutting through the bone by a Gigli's saw or surgical engine. The bone at the base of the flap must be broken. This method is the best when making large openings at the vertex, frontal, or parietal regions.

On exposure of the dura mater, the latter bulges into the wound, and cerebral pulsation is ill-marked if the intracranial pressure is much increased. The dura is opened crucially or by a flap, and the brain explored and any mass removed. Cysts and tuberculous foci can usually be shelled out with little hæmorrhage, but other tumours are ill-defined and require the brain substance to be cut.

The two-stage operation, in which the skull is opened one time and the brain explored subsequently, has the advantage of lessening shock, but the disadvantages of increasing the dangers of sepsis, and the bleeding from granulations.

The bone can be replaced if the fragment is large, and the wound drained for twenty-four hours.

If the operation is unsuccessful or incomplete, a hernia cerebri will probably form.

CEREBRAL LOCALIZATION.

The localization of function in different parts of the brain is shown by the disturbance which follows injury and disease.

LOCAL LESIONS may be: (1) Punctured wounds; (2) Depressed fractures; (3) Pressure of hæmorrhage or pus; (4) Pressure of scar tissue; (5) Abscess; (6) Tumour. These lesions may produce the following symptoms in different regions:—

UPPER LEFT FRONTAL LOBE.—Cerebral irritation, followed by dementia or insanity.

THIRD LEFT FRONTAL CONVOLUTION.—Motor aphasia. In left-handed people these two symptoms are associated with right-sided lesions.

ROLANDIC AREA.—Convulsions, or paralysis of the opposite leg, arm, or face, the centres being in this order from above downwards.

UPPER TEMPOROSPHENOIDAL LOBE.—Deafness in the opposite ear.

THE OCCIPITAL LOBE.—Temporary hemianopia, which is permanent if the angular gyrus is also affected.

CEREBELLUM.—Giddiness and staggering: the patient falls to the side of the lesion. Weakness of the arm and leg of the same side.

OTITIS MEDIA.

Varieties.—Catarrhal, acute purulent, and chronic purulent.

Causes.—Pharyngitis—Pharyngeal catarrh (spreads up the Eustachian tubes)—Tonsillitis—Scarlet fever and other fevers—Diphtheria—Adenoids.

Otitis Media, *continued.*

Symptoms.—Great pain on onset, relieved when the drum perforates.

Temperature raised to 100°–105° F.

Deafness—Usually partial only—Tuning-fork can be well heard if applied to the bones—Tuning-fork can be better heard in the affected ear when held to the vertex.

Tinnitus.

Purulent discharge from the meatus: only after the drum has perforated.

Signs.

BEFORE PERFORATION OF THE DRUM.—Drum is first red, and then has lost its lustre, and is abnormally bulged outwards. It does not move on swallowing or on politzerization.

AFTER PERFORATION.—On cleaning away pus from meatus, tympanum is seen to be perforated. In old cases the tympanum is destroyed, and bare bone or the ossicles may be seen or felt. On politzerization air escapes from the meatus if the Eustachian tube is patent.

Treatment of uncomplicated cases.

CATARRHAL OTITIS.—Usually associated with pharyngeal catarrh or adenoids. Treat the pharyngeal catarrh with astringent gargles or sprays, e.g., protargol gr. iv ad ℥j. Remove adenoids. Syringe meatus with warm boracic lotion. For acute pain of "earache," leech in front of the tragus, and hot sand-bags.

ACUTE PURULENT OTITIS.

BEFORE PERFORATION, or if the perforation is minute, artificial leeches in front and behind the ear. Free incision of the tympanic membrane from the centre downwards, followed by boracic irrigation. General treatment of the febrile condition.

AFTER PERFORATION with free discharge has taken place, douching meatus with 1–2000 biniodide of mercury. Dry meatus and instil boracic powder.

Complications of purulent otitis media—most commonly seen with the chronic cases.

EXTRACRANIAL.—Eczema of the meatus. Boils and deep inflammation of the meatus. Suppurative arthritis of temporo-maxillary joint.

CRANIAL.—Ankylosis or necrosis of the ossicles. Necrosis of parts of the temporal bone. Polypi (granulations) in place of the destroyed drum. Facial paralysis from pressure on the seventh nerve. Mastoiditis.

INTRACRANIAL COMPLICATIONS.—Subcranial abscess—Meningitis—Thrombosis of the lateral sinus—Cerebral abscess in temporo-sphenoidal lobe or cerebellum.

Acute Mastoiditis.

SYMPTOMS.—Intense pain of a constant character. Tenderness behind the ear over the mastoid process. Redness and œdema over the mastoid. Auricle is displaced forwards, outwards, and downwards. Eventually a fluctuating abscess forms behind the ear. General febrile symptoms : Temperature rises 102°–104° F. —A rigor may occur (but repeated rigors point to sinus thrombosis).

TREATMENT.

IN EARLY STAGE, before the diagnosis is certain : Leeches in front and behind the ear. Hot fomentations.

WHEN THE DIAGNOSIS IS CLEAR : Open and clear out the mastoid antrum. Incision curved behind the auricle. Gouge or drill over a point where the tangents above and behind the meatus cross. Antrum is reached a quarter to half an inch below bony surface. Remove all the bone between the antrum and the external meatus, protecting the seventh nerve by a probe passed from antrum to attic. Incise the cartilaginous meatus posteriorly. Sew back the two flaps to the pinna. Drain the whole cavity through the meatus.

Intracranial Complications of Otitis Media.

DIAGNOSIS —In all the following occur: Marked cerebral symptoms. Acute headache. Coma or delirium. Purulent discharge from the ear becomes scanty or ceases.

SINUS THROMBOSIS.—Repeated severe rigors. Temperature rises to 103°–105° F. but falls between the rigors. Patient is blue and collapsed. Tenderness or swelling over the internal jugular vein.

MENINGITIS.—Initial rigor, with continuous high temperature. Retraction of the head. Optic neuritis. Intolerance of light and sound. Rapidly developing coma.

SUBCRANIAL ABSCESS (pus between the dura and the petrous bone).—Severe headache. Gradual cerebral compression. Irregular fever. Absence of the signs characteristic of thrombosis, meningitis, or abscess.

CEREBRAL ABSCESS.—Temperature is normal or sub-normal. Constant headache, merging into coma. Slow pulse. Slow respiration—Cheyne-Stokes later. Optic neuritis and paralysis of pupil on affected side.

CEREBELLAR ABSCESS.—In addition to the above, may be nystagmus. Giddiness, with tendency to fall on affected side.

Treatment of Intracerebral Complications of Otitis.—First expose and ligature the internal jugular vein in the neck (if sinus thrombosis is clear). Trephine at a point one and a quarter inches behind meatus and a quarter of an inch above Reid's base line with a 1-in. trephine. This exposes the sinus and the dura above and below it. Open up and drain any subdural abscess. If thrombosis exists, open the sinus, clear out clot, and pack.

IF EVIDENCE POINTS TO AN ABSCESS, enlarge bone opening to a point three-quarters of an inch above the meatus, and explore the temporosphenoidal lobe. Enlarge bone opening downwards and backwards. Explore cerebellar hemisphere.

CHAPTER XXVII.

DISEASES OF THE LIPS AND JAWS.

THE LIPS.

Macrocheilia, or Thick Lips.

1. CONGENITAL.—Lymphangiectasis—Clear lymph-vesicles can be seen on the surface—Generally affects the lower lip.
   TREAT by excision of a wedge-shaped piece.
2. TUBERCULOUS.—In children and young adults—Generally the upper lip—May be associated with tuberculous disease of the nose—Fibrous and œdematous thickening round fissures.
3. SYPHILITIC.—Fibrous hypertrophy in tertiary disease.

Ulcers of the Lips.

1. SIMPLE.—Cracks and fissures result from cold in those with bad circulation. Chilblains and ulcerated chilblains arise in the same way.
2. HERPES.—Generally unilateral. Associated with catarrh or pneumonia. Crop of vesicles become pustular and then break.
3. TUBERCULOUS.—Either chronic indurated fissures causing macrocheilia; or definite lupus, with clear tubercles and destructive ulceration.
4. SYPHILITIC.
   PRIMARY CHANCRE.—Usually the upper lip. Caused by kissing person with secondary lesions of mouth, or using pipe or cup contaminated by such a patient. Flat ulcer on an infiltrated base. Skin more involved than the mucous membrane. Glands enlarged and massed together under the jaw (not so discrete as in genital chancre).
   SECONDARY.—Mucous tubercles and shallow, painful ulcers.
   TERTIARY.—Deep serpiginous ulceration destroying the lip—glands not enlarged. Or general fibrous hypertrophy (macrocheilia).
   INHERITED.—Cracks and fissured ulcers radiating from the angles of the mouth. Leave permanent scars and contraction.
5. MALIGNANT.
   EPITHELIOMA.—95 per cent are in men—generally clay-pipe smokers. Much commoner in lower than upper lip. Begins as crack, ulcer, or wart. Causes an ulcer or warty growth—indurated and everted margin, and indurated base. Submental and submaxillary glands become hard and enlarged comparatively late, i.e., after three to nine months.
   Much slower in its development than epithelioma of the tongue or fauces.

Diagnosis should always be made certain in doubtful cases by removal and microscopical examination.

TREATMENT.—Remove with at least half an inch margin. Dissect out submental and submaxillary glands from both sides, whether these are felt enlarged or not.

Death occurs from septic ulceration of the glands in the neck.

Tumours of the Lips.—Besides the ulcers given above, the following occur :—

WARTS.—Remove for fear of malignant growth, and examine with microscope.

NÆVI.—Dissect out or destroy by electrolysis.

CYSTS.—Caused by retention in the labial mucous glands. Small round fluid swellings containing glairy mucous fluid. Dissect out.

Hare Lip.—Congenital cleft in upper lip. Usually single and unilateral. Often double and bilateral. In simple cases the soft parts only are affected. In others the alveolus of the jaw or the palate may be cleft. When complete it extends into one nostril. Commoner on left side.

In all cases the nose is broad and flat. In double cases the bone between the clefts projects forward like a proboscis. Often associated with other deformities, e.g., talipes, etc. If the alveolus is involved, the cleft generally goes between the central and lateral incisor.

CAUSED by a failure of union of the internal nasal process with the maxillary process superficially, and the external nasal process more deeply.

EFFECTS.—Interference with suckling, and with speech. Deformity, which gets worse with age.

TREATMENT.—Operate from six weeks to three months.

FOR SINGLE HARE LIP.—Separate central parts of the lip from bone beneath. Pare the edges. Leave single or double flap of red margin, so that when sewn together there will be an allowance for contraction. Two deep stitches to take off tension. Many fine horsehair stitches to edges. Plaster strapping across the face to relieve tension. Put arms into splints. Feed by spoon, or, better, nasal tube. Allow to suck at end of one week.

FOR DOUBLE HARE LIP.

Treatment of os incisivum when projecting forwards: (1) Separate the lip from the bone ; remove os incisivum by cutting through base ; or (2) Refresh the anterior end of the nasal septum ; push back the proboscis and sew to the septum.

Treatment of cleft : Separate lip from the underlying bones. Pare both edges of both clefts. Sew outer margins of both clefts together below central part, which takes no part in margin. Leave a flap from red margin of each outer margin of each cleft.

CLEFT PALATE.

A congenital defect in the union of the two halves of the palate.

Degrees.—(1) Bifid uvula; (2) Cleft soft palate; (3) Soft and hard palate cleft as far forward as the anterior palatine foramina; (4) Including the alveolar border of the maxilla and lip, i.e., combined with a double hare lip.

Varieties.

1. Mesial—the cleft is in the mid-line, and the nasal septum is above and ununited to any part of the cleft.
2. Bilateral—the nasal septum grows down and divides the cleft into two.
3. Unilateral—the nasal septum unites with one side of the cleft. The cleft is usually on the left side.

Development of Alveolar Cleft Palate.[*]—In man the premaxillæ are usually rudimentary pieces of bone which are attached to the apex of the vomer. They take no part in the formation of the face, and bear no teeth. The maxillæ, on the other hand, send two processes inwards, which surround the premaxillæ; these are:—

1. The incisor process in front, which meets its fellow and bears the incisor teeth.
2. The palatine process, behind which forms the hard palate.

Between these two the anterior palatine foramen is left, in the inner wall of which the rudimentary premaxillæ remain.

In simple cleft palate, the palatine processes of the maxillæ are defective.

In alveolar cleft palate the incisor processes of the maxillæ are defective, and the premaxillæ grow forward into the face, as is the rule in lower animals. The alveolar cleft is then the space between the abnormal premaxilla and the undeveloped incisor process of the maxilla.

In the most extreme cases the premaxillæ bear all four incisor teeth, and there is a cleft on each side of them separating them from the maxillæ bearing the canines.

In less-developed cases the premaxillæ only bear one incisor, and the incisive process of the maxilla bears the other, and the cleft comes between the central and lateral incisors.

Appearances.—Through the cleft are seen the nasal septum and the turbinate bones. The width is greater in the more complete cases. The slope of the sides varies, being upwardly directed rather than horizontal.

Effects.—Suckling is impossible, and the infant must be spoon fed.

Defective speech is caused by the impossibility of closing the oral from the nasal cavities. Thus explosive sounds, e.g., the letters *T*, *D*, *P*, *B*, *F*, *V*, become *M* or *N*, and gutturals, e.g., *K* and *G*, become *H*.

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[*] *Prof.* Fawcett, *Bristol Medico-Chirurgical Transactions*, September, 1905.

Treatment.—Operative : Suitable for all cases up to twenty years.

OBTURATOR : A dental plate is fitted to fill up the gap ; but this never benefits the speech much, because it cannot act like a normal soft palate.

LANGENBECK'S, SMITH'S, OR THE LATE OPERATION.—Performed at or about the THIRD YEAR, when the soft parts have attained some thickness and firmness. The edges of the cleft are pared, and the mucoperiosteum lifted off the bone from the cleft outwards. The soft palate is completely cut from the hard palate bone at their junction, the soft palate remaining connected with the mucoperiosteal flaps. Any bands passing from the pharynx to the upper surface of the palate are snipped through. Lateral incisions are made through half the length of the soft palate up to the second premolar tooth to relieve tension. The two halves are sewn together ; silver wire is used for the mucoperiosteum of the hard palate, and horse-hair for the soft.

It is essential that the soft palate shall come together without any tension.

Gaps often occur in the hard palate, but are easily remedied by subsequent operations ; to get a sound and mobile soft palate is the chief object aimed at.

Long and careful speech education is required to correct the faulty articulation.

ADVANTAGES OF THE LATE OPERATION.—(1) The danger to life is negligible ; (2) The parts are firmer and thicker than in infancy ; (3) The cleft naturally diminishes within the first three years of life ; (4) The soft palate, by its separation from the hard, has the best chance of union and subsequent mobility.

DISADVANTAGES OF THE LATE OPERATION.—(1) That the parents are seldom satisfied to wait two or three years ; (2) That defective speech is acquired before the operation ; (3) Gaps often recur over the hard palate which require subsequent operations.

DAVIES-COLLEY'S, THE FLAP OR EARLY OPERATION.—Performed at the THIRD MONTH, i.e., before any teeth have erupted. A mucoperiosteal flap is cut from one side, the outer free margin of which lies outside the gums. This is turned over, the attached margin at the cleft acting as a hinge. The mucoperiosteum is raised from the opposite side as a flap, having its hinge outside over the gums. The first flap is turned over to the opposite side with the raw surface downwards, and stitched between the opposite flap and the bone. The soft palate is completed by a separate mucous membrane flap.

CLAIMS OF THE EARLY OPERATION.—(1) That the defect is closed long before the child has learned to speak badly ; (2) That a double layer of tissue is used to close the chief part of the gap ; (3) That there is no tension on the flaps thus made ; (4) That there is no interference with the muscles and nerves of the soft palate.

Cleft Palate—Treatment, *continued.*

Drawbacks to the Early Operation.—(1) The risk to life from asphyxia, hæmorrhage, and sepsis, when done so early; (2) The fragile nature of the tissues; (3) The smallness of the available operative space; (4) The large raw surfaces left in the mouth to heal by granulation; (5) The soft palate, not being separated from the hard, does not come together well, and is therefore likely to fail in union.

BROPHY'S OPERATION.—Performed at three months, in cases where the cleft is complete and combined with double hare lip. The os incisivum is removed, and the maxillæ are forcibly pressed together by partial fracture. They are wired in position through the alveolar processes, the edges of the cleft being pared.

Advantages.—The facial appearance is improved, and the cleft remedied by a bony bridge.

Disadvantage.—The danger to life, which has been considerable.

AFTER-TREATMENT.—Feed for one week by a nasal tube. It is wise to have educated the child to this before the operation. Remove the stitches from ten days to a fortnight.

THE JAWS.

Alveolar Abscess.—Results from a carious tooth. May open on the surface of the alveolus, externally, on the palate or into the antrum. Often causes periostitis, rarely necrosis.

Treat by removal of the tooth and opening the abscess.

Epulis.—A growth from the alveolar periosteum.

(1) Simple. Is a fibroid, firm and slow growing.

(2) Malignant. Is a myeloid sarcoma, soft, very vascular, easily bleeding and becoming ulcerated.

In each case the tumour grows from the sockets of carious or absent teeth. In the former, simple removal, in the latter, removal with a segment of bone surrounding it.

Epithelioma begins in the gums, or spreads from the cheek or tongue. It rapidly invades the underlying jaw.

Pyorrhœa Alveolaris (Riggs's disease).—A chronic suppuration between the roots of the teeth and the gums. Preceded and caused by a deposit of tartar. The pus forms pockets and sinuses beneath the gum margins. The gums retract from the teeth, and the teeth become loose. It may cause dyspepsia, a chronic sapræmia, and even chronic arthritis or synovitis.

TREATMENT.—Scaling teeth, slitting up sinuses, long-continued daily irrigation with antiseptics.

Necrosis of the Jaw.

CAUSES.—Dental caries and alveolar abscess—Compound fracture of the jaw—Injury and sepsis during teeth extraction—Phosphorus—Tertiary syphilis—Tubercle (rare)—Mercury poisoning—Osteomyelitis after exanthemata.

IN PHOSPHORUS JAW.—The disease starts round a carious tooth socket in those who work in the fumes of yellow phosphorus. Great swelling of chronic course results from a new periosteal bone formation. Sinuses open inside and outside the mouth. There is great fœtor, with a tendency to sapræmia, dyspepsia, and septic pneumonia. The sequestrum is grey and porous, and very long in separating.

TREATMENT is conducted on general principles.

Cysts of the Jaw.

SIMPLE DENTAL CYST.—Found in the neighbourhood of the roots of carious teeth, generally the upper first molars and bicuspids. Probably due to the irritation of an epithelial rudiment of the enamel organ. A painless swelling expands the bone, which presents egg-shell crackling. It contains mucoid material and some epithelial débris.

TREATMENT.—Removal of the tooth, with free opening of the cyst cavity.

EPITHELIAL ODONTOME, or fibrocystic disease of the jaw, usually affects the lower jaw, and in young people. It is caused by a proliferation of the epithelium of the tooth germ or enamel organ in the form of columns of cells resembling an epithelioma, but having a benign course and undergoing a cystic degeneration. A huge mass is formed, which expands the whole body of the jaw, the skeleton of which forms a bony framework for the cysts.

TREATMENT is by removal of a large part of the jaw, usually nearly half the mandible.

FOLLICULAR ODONTOME, or dentigerous cyst of the jaw, is also more common in the lower jaw. It is caused by an expansion of the follicle which contains the developing permanent tooth. Hence it is always associated with the absence of one of the adult teeth. The body of the jaw is expanded and thinned, the bone becoming like parchment. Inside the cavity is found the crown of the missing tooth.

TREATMENT by free opening and emptying.

MYELOID SARCOMA, when of central origin, may break down to form large blood-cysts. It also produces great expansion of the jaw.

CYSTS OF THE ANTRUM (see below).

Diseases of the Maxillary Antrum.

SUPPURATION.

CAUSES.—Carious teeth, especially the molars and premolars. Suppuration in the nose or accessory nasal sinuses.

SYMPTOMS.—Pain and tenderness, with neuralgia of the infra-orbital nerve. Intermittent discharge of pus from the nose. This may be seen to come from the middle meatus under the middle turbinate bone and above the inferior. It runs out when the patient holds his head forward, and trickles into the pharynx when he lies down. This is due to the fact that the opening into the nose is in the upper part of the antrum.

Diseases of the Maxillary Antrum—Suppuration, *continued.*

In Acute Cases the nasal aperture is usually blocked. The symptoms are more severe, and may merge into those of osteomyelitis.

Signs :—

Pressure Signs : (1) Nasal obstruction and blocking of the nasal duct; (2) Some exophthalmos; (3) Fullness of the palate, with irregularity of the teeth; (4) Swelling of the cheek.

Transillumination by an electric lamp in the mouth shows a darkness instead of the normal rosy colour under the eye.

Treatment : (1) Removal of offending tooth, and drainage through a metal tube inserted into this; (2) Free removal of the anterior wall under the cheek and of the nasal wall, and scraping out mucous membrane and polypi.

MUCOCELE, or cystic distention.

Causes.—A cystic distention of the glands of the mucous membrane, or the formation of a dental cyst.

Signs are merely those of slow painless distention, with atrophy of the anterior bony wall, which gives egg-shell crackling.

Treatment as in the last case.

INNOCENT TUMOURS.—Fibroma, odontoma, osteoma, cysts, and polypi.

MALIGNANT TUMOURS.—Sarcoma or carcinoma. Sarcoma is much the commoner. It causes a steady enlargement of the antrum, with signs which result from the bulging of its five surfaces: (1) The cheek; (2) The orbital surface, with exophthalmos; (3) The nasal surface, with nasal obstruction and epiphora, from blocking of the nasal duct; (4) The palate; (5) The zygomatic surface, producing swelling behind the jaw. Pain is very great and almost constant.

Treatment is by excision of the upper jaw.

Leontiasis Ossea.—Begins in young adults with no known cause. Consists in exuberant spongy exostoses growing from the cranial and facial bones, especially from the maxillæ, mandible, and nasals.

Produces hideous deformity, severe neuralgia, displacement of the eyes, and lastly pressure on the brain.

Treatment.—Early removal of the masses as they appear.

Inability to open the Mouth.

CAUSES : (1) Ankylosis of the joint, resulting from sepsis, tubercle, or osteo-arthritis; (2) Cicatricial contraction of the soft parts round the joint after injuries or operations; (3) Spasm of tetanus; (4) Reflex spasm from carious teeth, especially an unerupted wisdom; (5) Inflammatory swellings inside or outside the joint, e.g., mumps or tonsillitis; (6) Malignant tumours of the parotid or inside the mouth.

TREATMENT is only possible or desirable in the first two cases.

EXCISION OF THE CONDYLE is difficult, and often impracticable. An attempt may be made to interpose a vulcanite plate or some soft tissues between the bone surfaces to prevent bony union.

ESMARCH'S OPERATION.—Removal of a wedge-shaped piece of bone from the angle of the jaw on both sides, the apex of the wedge being at the alveolar border.

CHAPTER XXVIII.

AFFECTIONS OF THE NOSE.

Flattening of the Bridge of the Nose.—Destruction or impeded growth of the nasal septum.

CAUSES.—Injury, syphilis (usually congenital), tuberculous rhinitis (very rare).

TREATMENT.—Injections of hard paraffin melting at 105° F., by a special syringe under the skin.

Bone-grafting has now been superseded by the above.

Destruction of the External Nose.

CAUSES.—Syphilis, lupus, rodent ulcer, epithelioma, or traumatism.

TREATMENT.

INDIAN RHINOPLASTY.—New nose formed by a flap turned down from the forehead.

ITALIAN METHOD.—Flap formed from the arm and cut from the latter when it has firmly grown on the face.

FRENCH METHOD.—Cheek flaps.

BLOXAM'S OPERATION.—A finger is sewn to the root of the nose after the nail and ungual phalanx have been removed. The soft tissues are split and sewn to the cheeks. Finger is amputated a week later.

Foreign Bodies in the Nose.—Beads, peas, etc., introduced by children, or rhinoliths—calculi formed by the deposit of calcareous salts in chronic rhinitis.

SIGNS.—Nasal obstruction, offensive purulent discharge.

TREATMENT.—Removal either by syringing or by forceps under an anæsthetic.

Rhinitis.

ACUTE.

CATARRHAL.—Ordinary "cold in the head."

SEPTIC.—Associated with foreign bodies, septic wounds, and sinusitis.

GONORRHŒAL.—Spreads from the lachrymal sac.

DIPHTHERITIC.—Spreads forward from the pharynx.

CHRONIC.

SPECIFIC.—Lupus, spreads from the face or tear-duct. Syphilis, congenital (snuffles) or acquired.

HYPERTROPHIC.—Causing considerable intermittent or permanent engorgement and hypertrophy of the nasal mucous membrane, chiefly over the inferior turbinate bones.

Signs.—Unilateral or bilateral nasal obstruction. Altered tone to the voice. The inferior turbinate bone appears enlarged, and encroaches upon the inferior meatus of the nose. This enlargement quickly subsides on syringing with cocaine or adrenalin.

Treatment.—Sprays, simple or astringent (e.g., boracic acid or protargol, gr. j ad ℥j). Touching with the electro-cautery. Removal of a part of the turbinate bone.

Atrophic.—The mucous membrane is dry and shrivelled. The septum is often deflected. Pharyngitis and laryngitis are frequent accompaniments.

Treatment is unsatisfactory.

Ozæna.—Syphilis and tubercle. Young women. Nasal cavities are enlarged by an atrophy of their walls. Scabs and crusts cover the mucous surfaces. A penetrating nauseous odour is caused, which the patient cannot smell. Sometimes associated with sinus disease.

Treatment.—Copious alkaline douches, followed by oily sprays of liquid paraffin, menthol, etc.

Accessory Sinusitis.—The following sinuses open into the nose :—

Sphenoidal Posterior Ethmoidal Cells	Into the spheno-ethmoidal recess at the highest part of the nasal cavities.
Middle and Anterior Ethmoidal Cells Maxillary Antrum Frontal Sinus	Into the middle meatus of the hiatus semilunaris, under cover of the middle turbinate bone.

Nasal Duct.—Into the inferior meatus.

CAUSES.—Bacterial invasion after catarrh, foreign bodies, operations, exanthemata.

SYMPTOMS AND SIGNS.

Headache, which is either localized over the sinus, e.g., over the frontal, or general, owing to the proximity to the base of the brain.

Neuralgia.—From pressure on nerves, e.g., the supra-orbital by frontal disease.

Purulent Discharge from the nose, which, except in the case of the sphenoidal and posterior ethmoidal sinuses, comes from the middle meatus.

Rhinoscopy.—A mass of polypi and granulation tissue occupies the place of the middle turbinate bone. Bare bone can often be felt. The swelling of the soft parts does not diminish much by cocaine or adrenalin injections.

Radiography may show a shadow in a frontal, or even in a sphenoidal sinus.

Transillumination shows a shadow in the frontal sinus, the lamp being held in the angle of the orbit.

Accessory Sinusitis, *continued.*

DIFFERENTIAL DIAGNOSIS.—The antral and frontal sinusitis are recognized by the local tenderness, pain, and swelling, and by the evidence of transillumination.

The sphenoidal sinus may give a shadow by the X rays if it is full of pus.

TREATMENT.

For Frontal Sinusitis.—Removal of the anterior wall of the sinus, and draining into the nose.

For Ethmoidal and Sphenoidal Suppuration, an attempt may be made to get at the focus of disease through the nose, after clipping away the middle turbinate bone, or through the os planum of the ethmoid by an incision on the inner wall of the orbit.

MUCOCELE OF THE FRONTAL SINUS.—As in the case of the antrum, the frontal sinus may be distended by a mucous cyst. This causes expansion and thinning of its bony walls, which give an egg-shell crackling. A swelling appears on the forehead, and the eyeball becomes displaced downwards and outwards.

Treatment.—Removal of as much of the anterior bony wall as possible, and of all the mucous lining.

Nasal Polypi are of two different origins: (1) Inflammatory, and (2) Neoplastic.

1. MUCOUS POLYPI are merely œdematous granulations hanging from the surface of a diseased ethmoid bone, which is affected by rarefying osteitis or caries. They occur in young adults, and cause nasal obstruction, often bilateral. Grow from the middle and superior turbinate bones. They frequently undergo cystic degeneration from the development of cysts in the glands of the mucous membrane covering them. After local removal they recur within a few months.

   Treatment: (*a*) Removal by wire snare under cocaine; (*b*) Usually a thorough erasion of the lateral mass of the ethmoid or the opening of a suppurating sinus is required, so as to remove the diseased bone.

2. FIBROUS AND SARCOMATOUS POLYPI.—Consist in all gradations between fibromata and sarcomata, usually beginning as the former and ending as the latter. Occur in children and adolescents most commonly. Grow from the base of the skull and occupy the nasopharynx. Cause nasal obstruction, with sanious discharge.

   Pressure Signs may be: (1) Pushing down the velum palati and causing asphyxia; (2) Expanding the nasal cavities and producing a widening of the nasal bridge; (3) Pressing the eyeballs outwards; (4) Extending into the base of the brain.

   When they are Malignant, secondary growth may occur in the lymph-glands of the neck. In these cases the primary growth is probably a lymphosarcoma of the pharyngeal tonsils.

Treatment is only possible in the early stages.

1. Removal by a snare through the anterior nares.
2. Removal through the mouth after splitting the soft palate.
3. Removal from the face after turning up the soft parts and enlarging the anterior nares by temporary displacement of the maxillæ.

Adenoids.—A hyperplasia of the lymphadenoid tissue at the back and roof of the nasopharynx.

CAUSES.—Constitutional delicacy, which causes repeated nasal catarrh. Hygienic defects, e.g., insufficient ventilation. Want of exercise and a consequent slovenly method of breathing.

SIGNS AND SYMPTOMS.—A foliate and more or less symmetrical lymphoid mass fills up the nasopharynx. This can be seen by posterior rhinoscopy and felt by the finger-tip. The size varies from day to day, becoming much larger if the child has a cold. Mouth-breathing. The nasal passages are obstructed and the mouth kept habitually open. It becomes hard and parched. Snoring is marked at night. Discharge of sero-pus from the nostrils and nasopharynx.

RESULTS AND SEQUELÆ.

On Nose.—The nostrils become narrow and slit-like, from a falling in of the lateral cartilages. The vomer and septal cartilage become distorted, because the nasal cavities are not large enough for their growth.

On Mouth.—The palate is high, narrow, and arched. The teeth are arranged irregularly, being crowded together from insufficient palate growth. This is most noticeable after seven years, when the large permanent teeth are making their appearance.

On Ears.—The nasopharyngeal catarrh affects the Eustachian tubes, and causes chronic obstruction of these, with deafness, and frequent otitis media and its complications. It is by far the commonest cause of deafness.

On Trunk.—The chest, from the blocking of the air inlet through the nose, becomes either flat or pigeon-breasted. There is great liability to bronchitis and phthisis. Kyphosis of the spine accompanies this change.

On the Mind.—Mental torpor and stupidity occur from defective aeration of the nerve centres, and from the restless and bad sleep produced by the nasal obstruction. The gaping mouth, deafness, dribbling, all increase the appearance of feeble-mindedness.

TREATMENT.

Climate.—An equable, dry climate like that of Egypt will often cure a slight case.

Exercises.—Systematic " breathing exercises " will cure most cases of adenoids if persisted in for long enough, but in most cases this is quite impracticable.

Operation.—Removal by a curette under a general anæsthetic. Careful training in nose-breathing is very important afterwards.

Causes of Nasal Obstruction.

1. SWELLING OF THE MUCOUS MEMBRANE.
   Rhinitis: simple, tuberculous, or syphilitic.
2. OBSTRUCTION DUE TO THE SEPTUM.
   - (*a*) SPURS of cartilage and bone growing from the vomerine crest. Treat by cutting through the mucoperiosteum and sawing off the crest.
   - (*b*) DEVIATIONS of the septum. These may be traumatic in origin, or come from ill-development. Treat by submucous resection if there is no other primary cause of the obstruction.
   - (*c*) HÆMATOMA and abscess of the septum. Treat by incision.
3. DISEASES OF THE ETHMOID AND OTHER ACCESSORY SINUSES (*q.v.*).
4. DISEASES OF THE TURBINATE BONES.
   - (*a*) VASOMOTOR ENGORGEMENT.—Much reduced by cocaine or adrenalin injections. Treat by: (*i*) Hygienic measures; (*ii*) Iron and strychnine internally; (*iii*) Cautery of the inferior turbinate.
   - (*b*) NASAL CAVITIES ARE SMALL in proportion to the turbinates. Usually unsuited for treatment. Small parts of the front end of the inferior turbinate may be removed by scissors or punch forceps.
   - (*c*) ENLARGEMENT OF THE MIDDLE TURBINATE BONE.—Often associated with severe headache. Treat by removal by the scissors and forceps.
   - (*d*) MORIFORM HYPERTROPHY of the posterior end of the inferior turbinate. Remove by a snare.
5. BLOCKING OF THE CAVITY.—Foreign bodies, polypi, new growths, etc.
6. BLOCKING OF THE POSTERIOR NARES. — Adenoids. Fibrosarcomatous polypi.

Epistaxis.

CAUSES.—Traumatism, after operations. Fractured skull, etc. Ulcer on the septum. Varicose veins. Vasomotor disturbances: (*a*) In adolescents; (*b*) In adults with high blood-pressure. In certain blood conditions, e.g., hæmophilia.

TREATMENT—after examination to discover the cause.

1. Cauterize the bleeding spot if possible.
2. Plug the nasal cavity with gauze soaked in adrenalin.
3. Plugging the nares (anterior and posterior) is seldom required.
4. An inflating plug introduced into the nose and then inflated.
5. Calcium chloride or lactate in drachm doses by the mouth.

CHAPTER XXIX.

AFFECTIONS OF THE TONGUE.*

Congenital Malformations.

ABSENCE or ARRESTED DEVELOPMENT (very rare).

BIFID or SPLIT TONGUE may require suture for appearance' sake.

TONGUE-TIE, or ANKYLOGLOSSIA.—The frænum is too short, and so sucking and speaking are difficult. It is very rare, though often alleged. Careless division in spurious cases has often caused severe bleeding, septic ulcers, or dangerous tongue-swallowing.

TONGUE-SWALLOWING.—Excessive mobility allows the tongue to fall back, and may cause death by suffocation.

Hypertrophy of the Tongue.

1. MACROGLOSSIA is a condition of lymphangioma which is usually congenital, but sometimes acquired. Clear vesicles are seen on the surface, usually near the tip, and the whole tongue enlarges. Lymphatic cysts are formed beneath the mucous membrane, the papillæ hypertrophy, and there are also newly-formed blood-vessels and small round-celled infiltration. The organ is liable to recurrent attacks of glossitis, after each of which it is larger than before. It becomes so large that it cannot be retained in the mouth, and secondary changes take place in the lower jaw.

   TREATMENT by V-shaped excision.

2. SIMPLE MUSCULAR HYPERTROPHY may occur in idiots, or without any other lesion.

3. INFLAMMATORY HYPERTROPHY may be seen after any variety of glossitis, but most frequently after syphilitic.

Injuries of the Tongue.

Wounds are most commonly produced by the teeth, either in epilepsy or eclampsia, or by blows on the jaw when the tongue is protruding. Hæmorrhage is severe, but healing is rapid.

Punctured wounds, as by pipe stems, may have a foreign body in their depth, and this may give rise to severe secondary hæmorrhage, or to chronic inflammatory thickening.

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* Butlin and Spencer, "Diseases of the Tongue."

Acute Parenchymatous Glossitis occurs generally in adult men. Cold, mercurialism, and debility are predisposing causes.

Rapid painful swelling takes place, which makes speech and swallowing, or shutting the mouth, impossible.

Death may occur from asphyxia or sepsis, but recovery is the rule, with some superficial sloughing.

TREATMENT in bad cases is by free longitudinal incisions.

STREPTOCOCCAL GLOSSITIS begins in the neighbourhood of the submaxillary gland, and ends as a rapid cellulitis of the neck, with œdema of the glottis.

TREATMENT by antistreptococcal serum and tracheotomy.

STAPHYLOCOCCAL GLOSSITIS usually results from wounds and injuries. An indurated swelling forms, which develops into an abscess deep in the tongue substance.

TREATMENT.—Free opening in the mouth, or in deep cases through the mid-line of the neck above the hyoid.

MERCURIAL STOMATITIS is accompanied by foul breath, spongy gums, and superficial sloughing.

TREATMENT is by chlorate of potash, with astringent gargles.

GANGRENE may result from acute glossitis, or as an extension from cancrum oris. In the former case natural cure by the separation of the slough is the rule.

Superficial Glossitis occurs in many septic mouth conditions and in dyspepsia and gout.

SIMPLE OR DYSPEPTIC.—The tongue becomes indented by the teeth, and in bad dyspeptic cases it is raw and excoriated.

TREATMENT by chlorate of potash, and that for the dyspepsia.

HEMIGLOSSITIS is rare, and usually of nervous origin and accompanied by herpes.

APHTHOUS GLOSSITIS, OR THRUSH, occurs in infants from the growth of the *Oidium albicans*. The tongue and mouth become red, and then are covered with white patches, which consist of masses of the organism. It is very contagious among children.

TREAT by care of milk supply, cleaning the mouth with alkaline and boracic washes.

ERYTHEMA MIGRANS, or wandering rash, occurs rarely and in debilitated children. Red, smooth patches are seen, surrounded by a raised yellow border. The latter spreads like ringworm, and neighbouring rings may intersect. It causes itching and salivation, and is very chronic.

No special treatment is available.

HERPES is most common in neurotic women. It is very painful, leaves troublesome ulcers, and is prone to repeated recurrences.

TREATMENT by applications of cocaine or of carbolic acid.

Chronic Superficial Glossitis.

LEUCOPLAKIA OR LEUCOKERATOSIS.

ETIOLOGY.—Only occurs between twenty and sixty, and very rarely in women. Syphilis, smoking to excess, especially rough pipes, taking raw spirits and hot foods, gout, rheumatism, and dyspepsia, are all predisposing causes.

PATHOLOGY.—Papillæ disappear. A layer of small round cells occurs between the Malpighian layer and epidermis, and the latter becomes horny.

1. THE SMOOTH TONGUE OF SUPERFICIAL GLOSSITIS.—This is the simplest of these affections; the papillæ have disappeared, the epidermis is thin and horny, and the corium thick and vascular. The tongue is very tender and sensitive, and often feels stiff and uncomfortable.
2. SMOKERS' PATCH.—In the early stage is a smooth, raw, red area, but later becomes crusted or pearly grey from the development of the horny epithelium. Occurs on the dorsum near the mid-line opposite the pipe stem.
3. LEUCOMA is the name of the more generalized affection. Here there is a film which may be bluish-grey or thick and horny, extending over the whole tongue or on to the gums and cheeks. The underlying tissues become inflamed very often, and patches are shed, leaving raw and sore areas.
4. ICHTHYOSIS is similar to leucoma, but the papillæ are thick and hypertrophied, forming hard, rough, warty areas.

SYMPTOMS.—The tongue feels hard and dry, and thirst is prominent. Tenderness and rawness are felt, especially when the patch is denuded of its covering, when ulcers, warts, and fissures are apt to form.

COURSE.—Lasts many years and is practically incurable. It frequently gives rise to epithelioma.

TREATMENT.—Avoiding smoking, and sweet, sour, sharp, or strong articles of diet. Alkaline gargles, borax and glycerin, and also boracic or eucalyptus ointment, put on at night after carefully drying the tongue. Also chromic acid, gr. v. ad ℥j. Excision of limited patches, warts, ulcers, or fissures.

HYPERKERATOSIS (or Black Tongue).—Patch of epithelium in the mid-line in front of the circumvallate papillæ becomes dark or black, and the papillæ enlarge to form long waving hairs. The colour is due to micro-organisms.

No special treatment is required.

Simple Ulcers occur in superficial glossitis, and are very intractable when they become chronic.

PAINT with chromic acid, and EXCISE if they do not heal at once, lest cancer occur.

Herpetic Ulcers occur in children or young adults as an acute condition, with some malaise, as a vesicular eruption, followed by ulceration.

Chlorate of potash is the best TREATMENT.

Irritation Ulcers affect edges of the tongue adjacent to broken carious teeth or badly-fitting tooth-plates. Ragged and sloughy base and edges. No induration at base. Rapidly heal when tooth is removed. If left may be the starting-point of epithelioma.

Dyspeptic Ulcers.—Generally on the dorsum near the tip. Associated with a severe grade of dyspepsia. Shallow and indolent. No induration. Painful.

TREAT the dyspepsia, and paint ulcer with lunar caustic.

Mercurial Ulcers occur after the separation of superficial sloughs. with all the fœtor and other signs of stomatitis.

Tuberculous Ulcer.—Complicates phthisis or lupus. Especially near tip. Left by the breaking of an abscess. Shallow ulcer with sharp cut edges and no induration. The surface is pale and flabby. Very painful.

TREAT by excision under cocaine.

Syphilis of the Tongue.

PRIMARY.—As in lip chancre. Much matting and enlargement of glands. Much infiltration of the base, but not much induration.

SECONDARY.—Shallow, multiple painful small ulcers, especially round the edges and under surface. Mucous patches and condyloma (Hutchinson's wart).

TREAT by painting with chromic acid, gr. x ad ℥j, over ulcers.

TERTIARY.

1. CHRONIC SUPERFICIAL GLOSSITIS.
2. DIFFUSE FIBROUS THICKENING of the tongue. Chronic interstitial glossitis, generally combined with (1). Tongue is large and hard. Distorted by furrows and fissures. Caused by fibrous tissue drawing in the surface in folds and furrows.
3. GUMMA WITH ULCERATION.—Patient is generally about forty. Begins as a swelling in the tongue substance. In mid-line, far back, is common situation, or at the side. Breaks and discharges "gummy material." Quite painless ulcer is left. Base formed by tough slough (like wet wash-leather). Sharply-cut edges—"punched-out." No induration of the base. No fixation of the tongue. No enlargement of glands. Remains stationary a long time. Scar may give rise to epithelioma.

Simple Tumours of the Tongue and Floor of the Mouth.

SALIVARY GLAND TUMOURS may grow from the submaxillary, sublingual, or incisive glands. The majority are endotheliomata, and consist of endothelial columns and cartilage.

RANULA is an obstructive or retention cyst, usually of the sublingual, rarely of the incisive glands (see page 264).

SALIVARY CALCULUS, with surrounding inflammation, may be impacted in Wharton's duct and form a hard swelling.

DERMOID CYSTS are usually in the mid-line in the floor of the mouth, between the mylohyoid and geniohyoglossi. These are sequestration dermoids. They may be lateral near the angle of the jaw, in which case they are probably tubulo-dermoids from a branchial cleft. They form a firm, elastic, yellow swelling, which projects below the chin.

TREATMENT by excision through the mouth in most cases.

PARASITIC CYSTS.—Cysticercus and hydatid cysts are extremely rare.

LINGUAL TONSIL may be chronically enlarged or subject to a follicular abscess. In either case surgical treatment, excision or incision, will be required.

THYROGLOSSAL DUCT TUMOURS AND CYSTS. — These occur in two situations: (1) At the back of the tongue in the foramen cæcum, as a tumour or a cyst, which is red, vascular, and about the size of a pea or cherry, and has the structure of a thyroid adenoma; (2) In the substance of the tongue near the hyoid bone, in which case they form cysts lined by ciliated epithelium, and containing mucus.

BLOOD-CYSTS of the tongue are due to hæmorrhage taking place into these.

TREATMENT is by excision.

LIPOMA may be: (1) Congenital, when it is large and deep-seated; (2) Superficial and small, occurring in old people, and becoming polypoid; (3) Deep-seated, occurring in late life, between the tongue and floor of the mouth.

FIBROMA.—Either polypoid or deep-seated. Is painless and of very slow growth.

ANGEIOMA.—This may be arteriovenous, capillary, or cavernous, the last being the largest and most important. It is usually congenital.

EXCISION will be required to avoid ulceration and hæmorrhage.

PAPILLOMA occurs in children and adults as a simple new growth, and also in cases of superficial glossitis, when it is a precursor of cancer. It should always be removed.

Sarcoma of the Tongue is a very rare disease. It occurs in young adults as an elastic mass in the tongue substance. Its nature is seldom recognized until removal has been done. In some cases it is encapsuled and comparatively benign, but in others (lymphosarcoma) which begin in the lingual tonsil, malignancy is well marked.

Epithelioma of the Tongue.

Etiology.—Much commoner in men, i.e., about 85 per cent. Occurs generally between forty and sixty. Caused by rough pipe, much smoking, irritation ulcer, syphilitic scars, etc., and also the injudicious use of caustics.

BEGINS as :

1. WARTY OUTGROWTH, often in chronic glossitis. This is by far the most important method of origin.
2. AN ULCER at edge, at junction of middle and posterior third. Probably from a rough tooth. On the dorsum in glossitis or syphilis.
3. CRACK OR FISSURE, especially in chronic glossitis.
4. SUBMUCOUS INFILTRATION (very rare).
5. EXTENSION from tonsils or larynx.

Signs and Symptoms.—Usually begins on the anterior two-thirds, and towards the edges. Crateriform ulcer, or rarely a warty growth. Hard, everted (rolled-over) margins. Sides slope down to a ragged, foul base. Bleeds on touching. Profuse fœtid discharge and profuse salivation. Base of the ulcer is very indurated. Glands in the neck hard and enlarged after about six months.

LATER.—Pain is very marked ; often localized in the ear. Saliva constantly dribbles from the mouth. Tongue cannot be protruded owing to the involvement of intrinsic muscles. Glands in the neck form a huge mass.

Extension.—To the fauces and tonsils—Gums and lower jaw—Floor of the mouth.

Secondary Deposits.

GLANDULAR.—The lymphatic glands of the neck are invaded generally within the first six months of the disease, and often before that. The submental and submaxillary groups are involved first, with growths at the tip or sides of the tongue ; and the carotid group, extending from the skull to the sternum, is affected later. The most constant position is at the carotid bifurcation. Usually in the early stages only the glands of the same side as the growth are affected, but later those of the opposite side become involved.

VISCERAL.—The liver, lungs, heart, suprarenals, and other viscera are affected with extreme rarity.

Complications.

GLANDS IN NECK.—Form large cystic swellings. Break and form foul septic ulcers. Cause death by tracheal pressure, or by pressure dysphagia.

SEPTIC PNEUMONIA.

SECONDARY HÆMORRHAGE.

Course.—Patient generally dies within one year.

Diagnosis.

From OTHER ULCERS (*vide supra*).

From ACTINOMYCOSIS.—Pus, with characteristic granules.

From INFLAMMATION ROUND SALIVARY CALCULUS.—Although there may be great induration, there is no real fixation. An incision will reveal the stone.

Every ulcer, wart, or fissure which does not at once yield to local or specific treatment should be removed and examined for epithelioma.

Treatment.—Removal of tongue and glands, except in the following:

1. Base of tongue is so infiltrated on both sides that it cannot be moved.
2. Extensive involvement of the fauces.
3. Involvement of larynx.
4. Extensive involvement of jaw.
5. Glands fixed to great vessels and trachea.

Operations for Cancer of Tongue.

PRELIMINARY to all operations.—Remove carious teeth or stumps. Scale and clean the remaining teeth. Rub off all possible fur from the tongue with dilute antiseptics. Treat any dyspepsia. Spray mouth with 1–80 carbolic, or 1–2000 biniodide of mercury, or protargol and glycerin 1 per cent solution.

IN BAD CASES, i.e., where operation is a very extensive one, where there is a foul fungating mass, a preliminary injection of streptococcal serum may be given as a prophylactic.

1. WHITEHEAD'S OPERATION.

INDICATIONS.—Tongue must be able to be protruded.

METHOD.—Transfix tip by two threads. Split the tongue down the raphe. Cut through mucous membrane in floor of mouth. Cut through median part of tongue and tie the lingual artery. Cut through faucial pillars and root of tongue. Remove both halves if growth has deep induration or is near mid-line. Powder the stump with iodoform.

Glands, together with deep fascia, should be systematically dissected out from both carotid and submaxillary triangles, whether felt enlarged or not—a fortnight after excision of tongue in bad cases, but as preliminary to excision (when lingual artery can be tied), in ordinary cases.*

2. KOCHER'S, OR LATERAL EXTRABUCCAL OPERATION.

INDICATIONS.—When root of the tongue is involved—The tongue cannot be protruded—The jaw or fauces slightly involved.

METHOD.—Incision. Lobule of the ear to great cornu of hyoid bone. Along hyoid bone up to symphysis. Remove glands and tie lingual or the external carotid. Remove diseased alveolus if necessary. Cut through mylohyoid muscle. Bring tongue into the neck. Remove tongue back to the epiglottis, and down to the hyoid bone. Tie opposite vessels as they are cut. External incision closed with drainage.

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* This radical removal of fascia and lymphatics is Butlin's operation, and can be combined with either of the following procedures.

Operations for Cancer of Tongue, *continued.*

3. MEDIAN EXTRABUCCAL—SYME'S.

INDICATIONS.—Floor of the mouth involved. Disease spreading to symphysis menti.

METHOD.—Incision from margin of lower lip down over the chin to the middle of the hyoid bone. Two perforations through the jaw at the sides of mid-line. Cut the jaw into two halves, or remove diseased part of jaw. Draw the tongue out. Cut through with scissors from below backwards and upwards. Tie vessels one by one as they are met. Wire the two halves of the jaw together. Sew up the external incision.

IN REMOVAL OF THE WHOLE TONGUE BY ANY METHOD.

PRECAUTIONS :—

PRELIMINARY TRACHEOTOMY.—At the time of operation, or a few days previously. Use Hahn's tube surrounded by sponge, or a dilatable jacketed tube, or plug pharynx with a sponge.

Or LARYNGOTOMY at the time. Easier—quicker—Heals more rapidly afterwards.

Or PHARYNGEAL TUBAGE (Crile).—A rubber tube is passed into the upper part of the larynx, and packed round with gauze. The anæsthetic is given through this.

SECURE THE STUMP BY A THREAD fixed to the cheek to prevent asphyxia.

AFTER-TREATMENT.—Leave the tracheotomy tube in place two to seven days in bad cases. Powder the inside of mouth with aristol, or iodoform, and wash out frequently with Condy or 1–80 carbolic. If temperature rises, gives 30 cc. antistreptococcic serum. Feed by rectum for twenty-four hours, by œsophageal tube for six days.

CAUSES OF DEATH AFTER OPERATION.—Shock—Recurrent or secondary hæmorrhage—Asphyxia from falling back of the stump on the larynx—Septic pneumonia—Septicæmia.

OPERATION RESULTS.—Immediate mortality: intrabuccal operation, 7 per cent; extrabuccal operations, over 20 per cent. Cases alive at the end of three years without recurrence, 20 per cent.

RECURRENCE usually takes place in the stump within six months, or in the cervical glands at a later period.

PALLIATIVE TREATMENT.—Removal of carious teeth and keeping the mouth clean with antiseptics, e.g., hydrarg. bicyanide 1–1000.

FOR PAIN.—Insufflation with powder containing morphia or orthoform. Packing ulcers with iodoform gauze. Excision of the lingual nerve. Lastly, morphia by hypodermic injections.

FOR SALIVATION AND FŒTOR.—Atropine as an injection, and iodoform locally.

CHAPTER XXX.

AFFECTIONS OF THE ORAL GLANDS, PHARYNX, AND ŒSOPHAGUS.

AFFECTIONS OF THE SALIVARY GLANDS.

Parotitis.

Varieties.—(1) Epidemic; (2) Duct Infection; (3) Pyæmic.

EPIDEMIC PAROTITIS, OR MUMPS.—Is infectious, epidemic, and affects children. Both glands are affected by a parenchymatous inflammation, which never suppurates.

Coincident (or metastatic) inflammation of the genital organs: (1) Testis; (2) Breast, or; (3) Ovary. May occur in this order of frequency.

TREATMENT is medical.

ACUTE PAROTITIS FROM DUCT INFECTION.—This may be caused by any septic condition in the mouth, but it frequently follows operations on the abdomen where mouth-feeding has been stopped. It is, probably, due in the latter case to the septic state of the mouth with sordes on the lips and teeth, and also to the diminished salivary flow owing to the absence of food stimulus.

It may be simple or suppurative, usually the former.

PYÆMIC PAROTITIS is always suppurative, and is caused by a blood infection from any distant septic focus.

TREATMENT of this, and the last, is by fomentations, and then by incisions when fluctuation appears. The incisions should be parallel to the zygoma, so as to avoid injury to the facial nerve.

Inflammation of the other salivary glands is rare, and calls for no comment.

Salivary Calculus is common in Wharton's duct, but rare in Stenson's. It forms a hard swelling in the floor of the mouth, and may lead to salivary obstruction, or fistula. It has been mistaken for epithelioma.

TREATMENT.—Excision through the mucous membrane.

Salivary Obstruction may be due to: (1) Calculus; (2) Cicatrization; or (3) Injury. Rare, except in the submaxillary gland.

SYMPTOMS.—Painful swelling of the gland after meals.

TREATMENT.—By removal of the obstruction, or of the gland.

Salivary Fistula.—Is rare, except in Stenson's duct. Caused by operation wounds and injuries.

TREATMENT by passing sutures, or drainage tubes, from the intrabuccal opening to the fistula.

Tumours of the Salivary Glands.

RETENTION CYST OR RANULA is very common in the sublingual, and very rare in the other glands. Consists of a mucocele, which may be a pure retention cyst, or a new growth. Forms a bluish swelling in the floor of the mouth, and is always unilateral. Wharton's duct overlies it.

TREATMENT is by excision.

SIMPLE TUMOURS.—These, as well as malignant growths, occur commonly in the parotid, and rarely in the submaxillary glands. They are usually of a complicated structure, and are now regarded as ENDOTHELIOMATA. Simple fibroma or adenoma are rare. The tumours contain proliferating columns of endothelium which undergoes mucoid degeneration, and in which cartilage is commonly developed. Hence, various admixtures of adenoid, fibroid, chondroid, and mucoid tissues are found.

SIGNS.—A firm elastic or cystic swelling, which moves freely, and is usually limited by the zygoma, anterior margin of the masseter, sternomastoid, and angle of the jaw.

SYMPTOMS.—Usually painless, but unsightly. Quite slow in growth.

TREATMENT is by excision.

MALIGNANT TUMOURS.—These are usually endotheliomata which have become malignant, but primary sarcoma, or carcinoma, may also occur.

SIGNS.—Fixation of the tumour to the skin and underlying tissues. Facial paralysis from involvement of the facial nerve. Extension into the zygomatic fossa and encroachment upon the mouth and pharynx.

TREATMENT is by excision, but this is only possible if the case is seen early.

AFFECTIONS OF THE TONSILS.

Acute Tonsillitis.—According to the part involved this may be: (1) Follicular; (2) Parenchymatous; (3) Superficial. But this distinction is probably quite artificial and unnecessary.

FOLLICULAR TONSILLITIS is the usual form.

SIGNS.—Both tonsils become swollen and inflamed, and all their lacunæ filled with mucopus, which may also form a membrane over the surface.

SYMPTOMS.—Swallowing is very painful, and the glands enlarge under the jaw, and the temperature rises to 103–105° F.

CAUSES.—Sepsis, sewer-gas, contact with septic wounds (hospital throat), septicæmia, scarlet fever, rheumatism, chronic enlargement of the tonsils.

DIAGNOSIS must be made from scarlet fever by the absence of the rash.

TREATMENT.—Salicylate of soda pushed to deafness. Chlorate of potash gargles.

Peritonsillitis or Quinsy.—Is suppuration in or round the tonsil, usually the latter, occurring between the tonsil and its bed.

SIGNS.—Bright red swelling of the fauces and soft palate, usually much more marked one side than the other. The tonsil itself is often buried in the swollen faucial pillars. Inability to swallow is almost absolute, and the pain and constitutional disturbance are severe. If left alone it occupies about eight days before the abscess bursts, which it generally does through the soft palate or anterior faucial pillar. Rapid recovery follows. Fluctuation and pointing of the abscess can be seen and felt with difficulty, because the patient cannot open the mouth.

TREATMENT.—An incision directly a soft spot can be found. This generally has to be made through the soft palate, and runs parallel to the anterior faucial pillar.

Chronic Tonsillitis. Hypertrophy.

CAUSES.—Sepsis, cold, debility, insufficient exercise. A previous attack of acute tonsillitis makes recurrent attacks the rule, especially in adults, and after each of these the tonsils are larger than before.

STRUCTURE AND ASSOCIATED CONDITIONS.—The enlarged tonsils may: (1) Stand out as protuberant masses which almost touch one another; or, (2) They may lie deeply concealed within the faucial pillars. Above the tonsil and between the faucial pillars is a recess, the supratonsillar fossa, into which the upper follicles open. The large follicles show open mouths, or these may be plugged with bacterial and mucoid débris. A lobe runs down on to the lateral surface of the tongue. In children the pharyngeal tonsil, or adenoids, is generally also enlarged.

SYMPTOMS.—Thickness of speech. Mouth-breathing is common because of the adenoids. Liability to repeated acute attacks of tonsillitis is the most serious result. Also considerable debility may result from the chronic septic absorption.

TREATMENT.

THE GUILLOTINE is the routine method of treatment, gas being necessary for children, and cocaine for adults. In many cases of deeply-seated tonsils this method of removal is very inadequate, because all that part which lies deep to the faucial pillars is left, and subsequently causes a recurrence of the symptoms. This can be avoided in some cases by making traction on the tonsil with a vulsellum before driving home the guillotine.

ENUCLEATION is the ideal method, but always requires an anæsthetic. The finger is placed in the supratonsillar fossa, and the tonsil shelled out from its bed, and then twisted or cut off. Bleeding is not severe, because the vessels are torn rather than cut. The danger of wounding the internal carotid is quite mythical, since the vessel lies three-quarters of an inch away from the bed of the tonsil the other side of the pharyngeal wall.

Syphilis of the Tonsil may be: (1) Primary (very rare); (2) Secondary superficial ulcers, which are common; (3) Gummata, or late tertiary ulceration, which cause great scarring and deformity.

New Growths of the Tonsil.

VARIETIES.—

EPITHELIOMA, which usually begins in the fauces or tongue.

LYMPHOSARCOMA occurs at any age, but usually in young people, and is intensely malignant.

MIXED-CELLED SARCOMA is rarer.

SIGNS AND SYMPTOMS.—Rapid enlargement without pain or inflammation. In the case of epithelioma deep ulceration soon occurs. In that of a sarcoma the mass is elastic, soft, and encapsuled at first. In all the submaxillary cervical lymph-glands rapidly become enlarged. Dysphagia, asphyxia, or secondary hæmorrhage are the usual causes of death, which occurs within one year.

TREATMENT.—Removal through the neck with all the lymph-glands of that side that can be found. The incision is that of Kocher's tongue excision, and the external carotid is tied.

AFFECTIONS OF THE PHARYNX.

Acute Pharyngitis is caused by septic absorption, and often accompanies scarlet fever, septicæmia, rheumatism, and syphilis. Pain, swelling, and redness of the fauces and mucous membrane, with dysphagia. In septic cases, œdema of the glottis may occur and cause asphyxia.

TREATMENT.—Inhalation of steam impregnated with carbolic or balsamic vapours. Salicylates by mouth. Multiple incisions in cases threatened by asphyxia.

Chronic Pharyngitis is caused by smoking, drinking raw spirits, over-use of the voice, e.g., in clergymen and hawkers. The vessels become atonic and dilated, and nodules of lymphoid tissue become prominent. An excess of sticky mucous is secreted, which is tenacious and causes hawking and retching.

IN THE FOLLICULAR VARIETY the enlarged lymphoid nodules are prominent.

IN THE ATROPHIC VARIETY the mucous membrane is smooth and glazed, and may be covered by crusts.

TREATMENT.

AVOIDANCE of speaking, smoking, and drinking alcohol.

SPRAYS of tannic acid, perchloride of iron, or protargol, in glycerin, or oily sprays containing menthol, eucalyptus, etc.

GALVANO-CAUTERY POINT, to all the large lymph-follicles.

Post-pharyngeal Abscess.

ACUTE.—Due to septic infection through abrasions, e.g., swallowing a fish-bone. Caused by pyogenic organisms, and develops between the pharynx and the prevertebral fascia. May cause asphyxia by suddenly bursting into the larynx. Forms a tender, fluctuating swelling at the back of the throat.

TREATMENT.—Incision through the pharyngeal wall, care being taken that the pus does not enter the larynx.

CHRONIC.—Due to tuberculous disease of the upper cervical vertebræ. It forms with little pain, or inflammation, deep to the prevertebral fascia, behind which it tracks out into the neck.

TREATMENT.—Open by an incision behind the sternomastoid, before bursting or septic infection through the mouth has occurred.

Syphilis of the Pharynx is: (1) Secondary, in the form of superficial mucous patches, or snail-track ulcers; (2) Tertiary, in the form of submucous gummata and deep ulceration, which in the process of healing causes great deformity by tying down the soft palate to the pharyngeal wall.

Stenosis of the Pharynx is due either to cicatrization after scalds, or most commonly after syphilis. In the commonest type, the remains of the soft palate are so bound down to the posterior pharyngeal wall that the nasopharynx is shut off from the mouth. In the more serious type, the fauces and soft palate and posterior wall are contracted so as to constrict the passage from the mouth to the œsophagus.

TREATMENT.—Division of the stricture and subsequent passage of bougies. If this fails, œsophagostomy will be required in severe cases.

Epithelioma is the only primary growth of the pharynx, and as a primary growth is rare. Its appearance, course, and treatment are similar to those of the disease at the back of the tongue.

AFFECTIONS OF THE ŒSOPHAGUS.

Malformations.

CONGENITAL.

Fistulous communication with trachea.

Stricture at the cardiac orifice of the stomach.

ACQUIRED.

PRESSURE DIVERTICULA.—Caused by the yielding of the wall in the region of a branchial cleft. Found in adults about thirty. Forms a large pouch. Opens into posterior wall of gullet, just below junction with pharynx. Lies between gullet and spine, generally towards the left side. Causes dysphagia by pressure on the œsophagus.

TREAT by excision from left side of neck.

TRACTION DIVERTICULA.—Small pouches on anterior wall. Near tracheal bifurcation. Caused by traction of adherent glands. Produce no symptoms. May cause lodgement of a foreign body.

Affections of the Œsophagus, *continued.*

Foreign Bodies in the Œsophagus.—Coins, tooth-plates, pins swallowed accidentally by children or lunatics.

CHARACTERS OF FOREIGN BODY which are of importance.

SIZE, causing blocking at the entrance, with fatal dyspnœa from pressure on larynx.

IRREGULARITY, causing it to catch and be impacted in the wall.

SHARP POINTS, causing perforation.

RESULTS.

PASSAGE, per vias naturales, especially with small, round, smooth bodies.

IMPACTION, especially in the case of large or irregular bodies.

Position of impaction: Opposite the larynx (6 inches from teeth); opposite tracheal bifurcation (12 inches from teeth); at lower end (18 inches from teeth).

ULCERATION of the gullet, resulting in cellulitis of the neck, or mediastinitis; opening into the trachea; opening into a large vessel with fatal secondary hæmorrhage.

MIGRATION to a distance along fascial planes, in the case of pins or needles.

SYMPTOMS of foreign body in œsophagus—Dysphagia—Dyspnœa, especially if the neighbourhood of the larynx is involved.

LATE SYMPTOMS.—Pain from ulceration and inflammation—Hæmorrhage.

TREATMENT.

LOCATION BY: Inspection of fauces—Inspection by œsophagoscope—Digital exploration—Probang—X rays.

IF WITHIN REACH OF THE MOUTH (i.e., about twelve inches from the teeth).—Remove by: Fingers — Forceps — Horsehair probang—Coin-catcher.

IF IMPACTED IN THE NECK, remove by œsophagotomy on the left side.

IF IMPACTED IN THE THORAX, push down if the body is smooth. Remove through a gastrotomy wound if low down. Or remove through an œsophagotomy wound if high up.

Œsophageal Obstruction.

CAUSES.

EXTRINSIC: Tumours in neck (e.g., goitre)—Tumour in thorax (e.g., aneurysm) pressing on the gullet.

INTRINSIC: Spasm—Impacted foreign body—Fibrous stricture —Malignant stricture.

Spasm of the Œsophagus.—Symptom of hysteria—Usually in young neurotic women—Associated with the globus hystericus —No emaciation—No difficulty in passage of large bougie.

TREATMENT.—Cold douches, antispasmodics, etc.

Fibrous Stricture of the Œsophagus.—Usually in middle-aged men. At upper or lower ends of the gullet. Caused by the contraction of corrosive ulcers; possibly by syphilitic ulcers.

SYMPTOMS.—Dysphagia—Regurgitation of little altered food—Little or no bleeding on passing a sound—Very slow in its development.

TREATMENT.

PASSAGE OF TWO OR THREE SIZES OF BOUGIE once a week.

SYMONDS' TUBE: Passed on a whalebone guide. Retained, with a thread fixed to the cheek for periodical removal.

DIVISION OF THE STRICTURE, followed by bougies.

From the mouth: By a concealed knife cutting backwards. Only suitable for high strictures.

From the stomach: By forcible dilatation of stricture. Only possible for stricture of lowest end.

By string saw: Patient swallows a string. Lower end is pulled out of stomach. Pulled up and down.

ŒSOPHAGOSTOMY.—When the stricture is high up. Œsophagus is sewn into left side of neck.

GASTROSTOMY for worst cases.

Malignant Disease of the Œsophagus.—Patients over forty-five; usually men.

SITUATION AND VARIETY.

1. OPPOSITE THE CRICOID CARTILAGE—6–8 inches from the teeth.
2. OPPOSITE TRACHEAL BIFURCATION—12 inches from the teeth.

   In both of these the growth is an epithelioma. The epithelioma presents no cell nests.
3. AT THE CARDIAC EXTREMITY—18 inches from the teeth. Growth is a columnar-celled carcinoma.

EXTENSION AND COMPLICATIONS. — Early ulceration—Invasion of trachea, bronchi, great vessels—Metastatic growths in the glands.

SYMPTOMS.—Dysphagia. Inability to swallow solids and, later, liquids. Regurgitation of more or less altered food. This is not so marked as in fibrous stricture, because there is not time enough for dilatation above the growth. Pain and cough. Vomiting of blood, and bleeding on passage of a sound. Very rapid emaciation.

COMPLICATIONS of malignant disease of the œsophagus.—Septic pneumonia, from perforation of the air passages. Aphonia, from involvement of the larynx. Laryngeal paralysis, from involvement of the recurrent laryngeal nerves. Cellulitis, or mediastinitis.

PHYSICAL SIGNS.—Arrest of a bougie. Growth may be seen by an œsophagoscope. Auscultation detects a gurgle over the spine opposite the stricture on swallowing fluid.

COURSE.—Death from inanition, 6–12 months.

TREATMENT.—Symonds' tube (unsatisfactory). Gastrostomy.

Chapter XXXI.

AFFECTIONS OF THE NECK.

CYSTS OF THE NECK.

Congenital.—Often do not appear until adolescence.

1. DERMOIDS.
   - Under the Sternomastoid: (*a*) Branchial tubulodermoids. May open along anterior border of sternomastoid as branchial fistulæ. May become malignant—branchial carcinoma.
   - In Mid-line: (*b*) Ordinary sequestration dermoids or
   - (*c*) Thyroglossal Duct Cysts: From Thyroid duct—Foramen cæcum in tongue—Base of tongue or floor of the mouth—Behind the hyoid bone—In front of the larynx or trachea. Adenomata may arise from their walls in any situation. These may open as a median fistula. Generally in mid-line low down near sternum.
2. CYSTIC HYGROMA.—Large shapeless mass of dilated lymph-spaces. Whole of the side of the neck and face, and down into the thorax. Not limited by any anatomical boundaries.

Acquired.

SEBACEOUS CYSTS.—Fixed to the deep skin surface.

BURSAL CYSTS.—Over thyroid cartilage—Between hyoid and thyroid.

HYDROCELE OF THE NECK.—Probably a lymph cyst. Under the sternomastoid, and extend into posterior triangle.

BLOOD CYSTS.—Aneurysm—Venous varix—Cyst communicating with vein.

GLANDULAR CYSTS.—Thyroid: move with deglutition. Salivary: rare except in the floor of mouth.

MALIGNANT CYSTS.—From branchial dermoids—From breaking down epithelial glands—From endothelioma of carotid gland.

HYDATID CYSTS.

CUT THROAT.

Suicidal.—Generally from left to right—More severe on left side.

Homicidal—Vary in position and extent.

Varieties.

1. Those not opening pharynx or air passages. Nothing special.
2. Those involving air passages.—Above hyoid—Through thyrohyoid space—Into larynx—Into trachea.

Primary Effects.

ABOVE HYOID.—Wound of lingual and facial vessels. Injury of root of tongue. Danger of asphyxia from tongue falling over larynx.

THYROHYOID SPACE.—Wounds of lingual, facial, and superior thyroid vessels over larynx. Injury of epiglottis. Bleeding into larynx.

LEVEL of LARYNX.—Injury to the vocal cords or lobes of thyroid gland.

BELOW LARYNX.—Hæmorrhage from thyroid gland and vessels—Trachea opened—Asphyxia from displacement of the cut ends or from bleeding into the trachea—Wounds of great vessels, with fatal hæmorrhage—Air drawn into veins—Division of recurrent laryngeal nerve.

Remote Effects.—Septicæmia or pyæmia—Cellulitis leading to mediastinitis—Œdema of glottis—Secondary hæmorrhage—Tracheitis, bronchitis, septic pneumonia—Passage of food into trachea—Surgical emphysema.

Treatment.—Arrest the hæmorrhage. Sew up, with free drainage. If larynx is opened, close it and perform tracheotomy. If trachea is opened, insert tube. If wound is above larynx, sew it up, drain, and perform tracheotomy.

Sequelæ to Cut Throat.—Aerial fistula, generally in the thyrohyoid space. Pharyngeal fistula. Laryngeal or tracheal stenosis. Aphonia from injury of larynx or recurrent nerve.

DISEASES OF THE THYROID GLAND.*

Atrophy is found in three conditions.

SENILE ATROPHY.—When it causes no symptoms.

MYXŒDEMA.—The epithelial tissues disappear partly or entirely, and what remains is only connective tissue.

CRETINISM.—The gland is either (1) Congenitally absent; (2) Represented by a mere connective-tissue rudiment; or (3) Present as a goitre, with few or no epithelial elements. It may be sporadic or endemic, the latter being common in districts where endemic goitres occur.

In both myxœdema and cretinism there occur:

1. CHANGES IN THE SUBCUTANEOUS TISSUES.—Great increase of fat and mucoid tissue. The hair is brittle and falls out. Supraclavicular fatty masses are very conspicuous.
2. CHANGES IN THE NERVOUS SYSTEM.—Amentia or dementia are prominent. In the case of cretinism a state of childishness or imbecility continues throughout life unless the case is treated.
3. CHANGES IN THE SKELETON.—The terminal phalanges become broad and spatulous. In cretinism growth is arrested, so that the stature of the adult is that of a child of about six.

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* James Berry, "Diseases of the Thyroid Gland.

Thyroid Gland, Atrophy—Cretinism, *continued.*

4. CHANGES IN THE GENITAL ORGANS.—The cretin has undeveloped sexual glands, undescended testes, and no secondary sexual characters. In myxœdema sexual feeling and power are lost.

Hypertrophy (apart from parenchymatous goitre) is found in the following :—

1. Of a portion of gland when the rest has been removed.
2. During menstruation (probably merely a hyperplasia) at puberty, during pregnancy, after sexual excitement.

GOITRE.

Classification.

PARENCHYMATOUS.—Uniform enlargement. The common form in young patients. It rarely begins after twenty-five.

CYSTIC.—(1) Distention of single vesicles; (2) Liquefaction of adenomata.

ADENOMATA.—Encapsuled masses in the gland substance: (1) Fœtal, consisting of solid epithelial columns with no colloid; (2) Cystic, consisting of ordinary thyroid tissue, many of the vesicles being dilated to form cysts.

FIBROUS.

EXOPHTHALMIC.

MALIGNANT.

Etiology.—It is usually endemic. The localities most affected are in Switzerland, mountainous districts of France and Italy, Derbyshire and Gloucestershire in England. It is associated with a limestone geological formation, which probably causes some peculiarity of the drinking water. Possibly this consists in the absence of iodine or iodides.

Physical Signs.

MOBILITY.—It moves up and down in swallowing. It cannot be separated laterally from the trachea. This alone usually suffices for the diagnosis, but :

1. OTHER CERVICAL SWELLINGS WHICH MOVE on deglutition are : Lymph-glands adherent to the trachea ; abscesses or malignant growths connected with larynx, trachea, or œsophagus ; subhyoid bursæ ; thyroglossal cysts.
2. GOITRES WHICH DO NOT MOVE with deglutition are : Very large masses which cannot pass through the thoracic inlet. Goitres which are fixed by malignant or inflammatory adhesions.

SHAPE AND SIZE.—Parenchymatous goitres may be horse-shoe shaped, but others are usually oval. The size is anything up to that of a man's head, in which case it hangs over the sternum.

POSITION.—Usually below the larynx. In the mid-line or lateral. Sometimes substernal, rarely high up on a level with the great hyoid cornu. Pushes the sternomastoid muscle outwards.

Relation to Vessels.—The common carotid is pushed backwards and outwards, whilst the internal jugular vein becomes spread over its surface, being tied to it by the thyroid veins. In malignant goitre the vessels become surrounded without much displacement.

Relation to the Sternum.—Occasionally substernal goitres descend as low as the arch of the aorta.

PULSATION.—Thyroid tumours often present a pulsation, which is either (1) communicated from the carotid, or (2) is that of the thyroid arteries, or (3) is due to the vascular nature of the growth, especially in Graves' disease. A bruit is often heard—loud and rasping—in these cases.

CONSISTENCE.—Soft when parenchymatous. Firm and elastic when adenomatous or cystic. Hard when fibrous or calcified.

PRESSURE EFFECTS.

On Veins.—The jugular veins become engorged, but œdema and cyanosis are only seen in malignant or inflamed growths.

On Nerves.—The recurrent laryngeal, sympathetic, cervical, and brachial nerves may be involved, in the order of frequency, but marked nerve involvement is a grave sign of malignancy.

On the Trachea.—This may be (1) displaced laterally, (2) kinked, or (3) compressed laterally so that its cavity is reduced to a chink. This is the common cause of dyspnœa.

On the Œsophagus.—Very rarely a posterior thyroid growth may cause displacement and compression of the gullet.

Dyspnœa.—This is practically the only symptom produced by goitres. It arises in several ways, pressure on the trachea being far the commonest.

1. TRACHEAL PRESSURE.—The trachea is displaced laterally, or sometimes backwards (by a substernal goitre). Its lumen is narrowed by (*a*) kinking, (*b*) unilateral pressure, (*c*) bilateral pressure. It becomes soft and yielding after long pressure, but not actually atrophied.
2. BY PRESSURE ON THE RECURRENT LARYNGEAL NERVES.—This is very rare, as shown by the infrequence of aphonia, combined with dyspnœa.
3. BY EXTENSION OF THE GROWTH INTO THE AIR-PASSAGES.—This only occurs in malignant disease.
4. BY RUPTURE OF A CYST OR ABSCESS INTO THE TRACHEA.
5. BY CAUSING ŒDEMA GLOTTIDIS.—Only in inflamed or malignant goitres.
6. BY SWELLING OF THE MUCOUS MEMBRANE of the air-passages. This occurs when the patient catches cold or contracts bronchitis.

Goitre—Dyspnœa, *continued.*

INCIDENCE OF DYSPNŒA IN GOITRE.—It occurs most often in patients between the age of puberty and twenty, in parenchymatous, bilateral, and substernal goitres.

ITS SUDDEN APPEARANCE may be due to hæmorrhage into a cyst, or the rapid enlargement of cystic cavities.

A BRASSY COUGH generally accompanies the dyspnœa, and is due to the tracheal affection.

Cystic Goitre.

OCCURS in middle-aged and old people.

MODE OF ORIGIN.—(1) By distention of normal vesicles and fusion with one another; (2) By liquefaction of soft adenoma, the capsule of which forms the eventual cyst wall. This is much the commonest origin; (3) By hæmorrhage into an adenoma.

VARIETIES.—Single, generally lateral; multiple, generally bilateral.

CYST WALL.—Thin, with a delicate epithelial lining in small cysts. Thick, fibrous, or calcified in large ones.

CONTENTS.—Viscid fluid, containing two-thirds colloid albumin. Masses of calcified tissue are common in very old cysts.

HÆMORRHAGE into the cyst cavity is very common, and may result from an injury, or very often follows tapping. It is caused by the delicacy of the vascular adenoma. It may result in urgent dyspnœa.

INTRACYSTIC GROWTHS.—True papillomata growing in the cyst wall are rare, and show a tendency to malignancy.

Hydatid Cysts.—Over twenty cases have been recorded. A cyst forms rapidly in a young patient. Pressure signs are well marked, and atrophy of the gland is produced. Death may be caused by pressure on the trachea or by bursting into the air-passages. Suppuration is frequent.

TREATMENT by enucleation, which is usually only partial.

Acute Inflammation.

CAUSES.—Operation on, or injection of a goitre. Infective fevers, typhoid, rheumatism, pyæmia, etc.

SYMPTOMS.—The ordinary signs of inflammation, with febrile symptoms. Dyspnœa is caused by pressure on the trachea, or if it already exists it is much increased. Dysphagia, with great pain, are prominent.

EXTENSION and suppuration may cause the abscess to burst: (1) Externally; (2) Into the cellular tissue of the neck; (3) Into the mediastinum; (4) Into the trachea; (5) Into the pharynx or œsophagus. In all but the first and last, death rapidly follows.

DIAGNOSIS from:

SUDDEN BLEEDING into a cyst or adenoma.

MALIGNANT DISEASE when growing rapidly, and causing much pain.

In both these there is an absence of febrile symptoms.

Inflammation of Lymph-glands, which do not move with the larynx.

Abscesses connected with the larynx.

TREATMENT.—Hot fomentations, opening abscess as early as possible.

Chronic Inflammation.—A rare condition of idiopathic chronic inflammation occurs. The thyroid becomes moderately large and very hard in a few months. Severe dyspnœa, aphonia, and pain are caused. It becomes attached densely to skin, great vessels, and trachea. Its removal is very difficult owing to these close attachments. It is generally regarded as a sarcoma, but consists of simple inflammatory tissue.

Tubercle and Syphilis.—These both occur with extreme rarity. In the latter disease gummata form in the thyroid and cause severe dyspnœa.

Exophthalmic Goitre.

ETIOLOGY.—Females form 90 per cent of the cases, and the age incidence is the early periods of sexual activity (fifteen to thirty). The local occurrence bears no relation to endemic goitre.

ANATOMY.—The gland is uniformly but not very greatly enlarged. It is of smooth surface and firm fleshy texture. The blood-vessels are neither large nor conspicuous. Microscopically the tissue shows great epithelial proliferation and diminution or absence of colloid material. The thymus gland is often enlarged.

PATHOLOGY.—The disease is due to: (1) Increased thyroid activity; (2) Perverted thyroid activity; (3) Increased absorption of thyroid secretion. Evidence of causative disease of the central nervous system or sympathetic is quite absent.

SYMPTOMS.

Cardinal: (1) Uniform and slight thyroid enlargement; (2) Prominence of the eyeballs, with delay in the descent of the upper lid when shutting the eyes; (3) Tachycardia, with attacks of palpitation.

Accessory: (4) Nervous symptoms, tremor, excitability, and attacks of acute mania; (5) Sweating; (6) Dyspnœa; (7) Diarrhœa; (8) Glycosuria; (9) Patchy pigmentation; (10) Local fatty swellings, especially on the abdominal wall; (11) Slight elevation of temperature.

Occasionally the goitre or the exophthalmos may be absent. The exophthalmos is due to an accumulation of fat in the orbits and to spasm of the unstriped muscle fibres which lie at the back of the eyeball. The dyspnœa is due to cardiovascular changes, and is not accompanied by stridor.

TREATMENT.

Medicinal.—Bromides, digitalis, and belladonna are useful. Iodine, iodides, and thyroid preparations are harmful. Moebius' serum, obtained from animals from whom the thyroid has been removed, is the most active remedy.

Exophthalmic Goitre—Treatment, *continued.*

SURGICAL.

PARTIAL THYROIDECTOMY has a mortality of 12 per cent, and in only about 30 per cent of cases does a cure result. The patients die from shock or from acute thyroidism.

ARTERIAL LIGATURE. — Both superior and one inferior arteries are tied, and some improvement results.

SYMPATHECTOMY.—Removal of both cervical sympathetic cords, including the superior ganglia. The mortality is high (12 per cent), and the results are uncertain.

The operations should be performed under local anæsthesia.

CONCLUSIONS.—The disease in most cases tends to spontaneous recovery. About 20 per cent die when no operation is performed. Over 12 per cent die when thyroidectomy has been done, and the proportion of recoveries is only a little higher than those treated medically. Therefore operation should be reserved for cases in which the disease is progressing in spite of general and drug treatment.

Malignant Disease.

ETIOLOGY.—Rarely occurs before forty. Affects both sexes equally. Often develops in a simple goitre.

STRUCTURE.—Alveolar carcinoma and sarcoma, either round- or spindle-celled, are of about equal frequency. Sarcoma is rather more likely to form localized swellings of rapid growth, and carcinoma general enlargement of slower growth; but usually the distinction is impossible.

SYMPTOMS AND SIGNS.—Special signs of a goitre being malignant are: (1) Hardness, with bossy outline; (2) Evidence of early nerve involvement, unilateral vocal cord paralysis, shooting pains up the neck and down the arm, contraction of the pupil and palpebral fissure; (3) Involvement of vessels. The carotid, instead of being displaced, is surrounded by the growth, and cyanosis with œdema of the face may occur late in the case from obliteration of the veins; (4) Penetration of the trachea, and much more rarely the pharynx, by the growth; (5) Great fixity to the surrounding structures. The skin and lymph-glands are, however, rarely involved.

METASTASIS occurs in the bones and the lungs. In the former case pulsating tumours are formed, especially on the cranial bones.

DEATH occurs in three months to two years.

CAUSES: (1) Dyspnœa; (2) Penetration of the trachea, with septic pneumonia; (3) Œdema glottidis; (4) Hæmorrhage into the growth, with sudden dyspnœa.

PAPILLIFEROUS ADENOMATA have only a limited malignancy. That is, they are very liable to recur, but at first do not infiltrate *the tissues* or cause metastases.

TREATMENT.

RADICAL.—Is only possible in a small proportion of cases; often involves resection of the great vessels or trachea. Operative mortality is 35 per cent, and survival for three years only 6 per cent.

PALLIATIVE.—Local removal of masses which compress the trachea.

Tracheotomy. This is rarely possible below the tumour. Often it has to be done through the growth. Generally it should be done above the growth and a long Koenig's tube, four to 5 inches long, passed down beyond it.

Treatment of Innocent Goitre.

GENERAL.—Removal of the patient from goitrous district.

Iodine and iodides (tr. iodi ♏v, pot. iod. gr. iv, t.d.s.), thyroid extract (gr. v, t.d.s.).

This drug treatment only acts in parenchymatous cases in young patients. It has no effect on cysts, adenomata, or fibroid goitres. Marked diminution will occur in one month or not at all.

Local applications—iodine and mercuric iodide ointment—may be applied to the goitre, but the effect is very slight.

INDICATIONS FOR OPERATION.—Dyspnœa in all cases. Cysts or adenomata. Failure of medicinal treatment in diffuse goitre.

PRECAUTIONS.—Use of local anæsthetic in all cases where dyspnœa is well marked; 2 per cent solutions of eucaine, or of 1 per cent solution of novocaine, are the best. Performance of the operation in the position of greatest ease as regards breathing.

OPERATIVE.

EXTIRPATION.—The operation of choice in diffuse goitre. One half or more of the gland is removed in its capsule. Skin incision, along anterior sternomastoid border, or a transverse incision for low, prominent goitres, this incision leaving a less prominent scar. Infrahyoid muscles are drawn outwards, or cut. The lateral and inferior veins are tied first, then the superior vessels, and lastly the inferior artery.

Special care is to be taken of (1) the internal jugular vein, as it lies expanded over the goitre, and (2) the recurrent laryngeal nerve, as it lies behind and in very close connection with the tumour below the cricoid cartilage.

Divide the isthmus and remove the lobe. Drain for twenty-four hours.

RESECTION is similar to the above, but the gland is cut through, so as to leave behind the part so intimately connected with the recurrent laryngeal nerve between the cricoid cartilage and œsophagus. Both lobes may be removed in this manner.

ENUCLEATION is the operation for cysts and adenomata, which are shelled out from a capsule formed by thinned gland substance.

Treatment of Innocent Goitre—Operative, *continued.*

OBSOLETE OPERATIONS.

TAPPING CYSTS.—Is only rarely curative, and often causes severe intracystic hæmorrhage.

INJECTION of iodine and other solutions for parenchymatous and cystic goitres is uncertain in its results, and there is great danger of causing severe inflammation.

LIGATURE OF THE VESSELS, if done thoroughly, is the most difficult part of extirpation. It is only used in exophthalmic cases.

DIVISION OF THE ISTHMUS sometimes relieves dyspnœa and causes shrinkage. This is due to escape of thyroid secretion, and not to a mechanical release of tracheal constriction.

ACCIDENTS which may occur during thyroid operations.

SUDDEN DEATH from dyspnœa occurs in cases where severe dyspnœa is present at the outset. Due to traction on, or pressure on, the trachea, the general anæsthetic, spasm of the glottis, or traction on the nerves in the neck. It is most liable to occur when the tumour is dragged on or turned out of its bed. It is best avoided by never giving a general anæsthetic in cases in which dyspnœa is severe.

PRIMARY HÆMORRHAGE.—This is more likely to be severe in enucleation operations. In extirpation all the chief vessels should be tied before they are cut. It is chiefly venous bleeding that gives trouble.

INJURY OF THE RECURRENT LARYNGEAL NERVE.—Causes unilateral vocal paralysis.

Injury of the sympathetic or vagus.—The latter may cause death.

Injury to the trachea, pleura, œsophagus, or pharynx.

Collapse of the flattened trachea. This is very rare, but may require tracheotomy.

ACCIDENTS WHICH MAY OCCUR AFTER THYROID OPERATIONS.

RECURRENT HÆMORRHAGE.—Specially after enucleations and if adrenalin solution has been used with a local anæsthetic. It may cause death from hæmorrhage or from dyspnœa. It is a wise precaution to let patient come partly round before sewing up.

SEPSIS, leading to cellulitis or mediastinitis, or to a chronic sinus.

RESTLESSNESS AND RAPIDITY OF PULSE AND RESPIRATION.—This is most often seen in operations for Graves' disease, next in parenchymatous goitres. It is probably due to rapid absorption of thyroid secretion. It is usually fatal. It is best avoided by careful handling of the tumour, free irrigation of the wound with water, and drainage.

LATE VOCAL PARALYSIS, due to involvement of the recurrent nerve in scar tissue.

TETANY.—Very rare. Probably due to removal of the parathyroid glands.

REMOTE COMPLICATIONS. Only seen after total thyroid removal.

CACHEXIA STRUMIPRIVA.—Symptoms develop two or three months after operation. Fatigue is complained of, and the limbs are heavy and indolent. The hands become swollen and clumsy, and all fine movements impossible. Skin is pale and much swollen, especially over the eyelids, forehead, cheeks, and tongue, but there is no pitting on pressure. The facial expression is dull and vacant. Cerebration is very slow, and memory is bad. Speech is slow and laboured. The skin becomes dry and scaly, and the hair falls out. The temperature is subnormal, and there is great sensitiveness to cold. If the patient is immature, growth and sexual development are arrested. In adults sexual functions cease.

TREATMENT.—Administration of thyroid extract.

CHAPTER XXXII.

DISEASES OF THE AIR-PASSAGES AND CHEST.

FOREIGN BODIES IN THE AIR-PASSAGES.

In Nasal Passages.—Common in children. Unilateral purulent discharge.

TREATMENT.—Syringing through sound nostril. Removal under an anæsthetic.

At the Rima Glottidis.—Food mass, or large foreign body. Rapid asphyxia and death.

TREATMENT. — Removal by the finger from the mouth, or immediate laryngotomy.

In the Larynx.—Small, round, foreign bodies. Produce urgent dyspnœa; croupy cough. Complete obstruction occurs later from œdema.

TREATMENT.—Remove by laryngeal forceps, laryngotomy, or thyrotomy.

In the Trachea.—Attacks of spasmodic cough and dyspnœa caused by the foreign body impacting against the larynx. Tracheitis, with cough and expectoration.

TREATMENT.—Low tracheotomy with removal; or stitch open the tracheal wound and wait for the foreign body to be coughed up.

In a Bronchus.—Smooth, round, heavy bodies, e.g., marble or intubation tube. Generally into the right bronchus, because: It is more in a line with the trachea; the septum between the bronchi lies to the left of the middle line; the right bronchus is larger than the left in the proportion of five to four.

SYMPTOMS.—Short attack of spasmodic dyspnœa, due to the passage through the larynx. Collapse of the corresponding lung, due to the air escaping more easily than it enters. Produces:—(1) Dullness and absence of breath sounds. (2) Unilateral bronchitis; bronchiectasis; (3) Abscess of the lung, or pneumonia; (4) Empyema.

RESULTS.—Death from one of the above. Expulsion with pus from an abscess by violent coughing. Expulsion with the pus of an empyema.

TREATMENT.—Removal through a bronchoscope; removal through a low tracheotomy; removal through the posterior chest wall.

DISEASES OF THE LARYNX.

Inflammatory Œdema.—Œdema glottidis, or œdematous laryngitis.

CAUSES.

1. Direct Injury of the larynx, e.g., by scalds, corrosives, or wounds.
2. Foreign Body impacted in the larynx.
3. Secondary to : (*a*) Any inflammatory disease of the larynx ; (*b*) Cellulitis of the neck (Ludwig's angina); (*d*) Acute glossitis ; (*e*) Retropharyngeal abscess.

ANATOMY.—Swelling and œdema of :—aryteno-epiglottidean folds ; inter-arytenoid fold ; epiglottis, which becomes folded on itself ; false vocal cords. Rima glottidis becomes partially or totally occluded.

SYMPTOMS AND SIGNS.—Intense inspiratory dyspnœa. Epiglottis and folds above the larynx are seen and felt to be swollen, red, and œdematous.

TREATMENT.

In Slight Cases : Scarification of the swollen mucous membrane. Ice compresses externally.

In Bad Cases.—Tracheotomy.

Syphilis of the Larynx.

IN THE SECONDARY STAGE.—Mucous tubercles. Superficial ulceration. Producing hoarseness and aphonia.

IN THE TERTIARY STAGE.—Gummatous infiltration, and deep destructive ulceration affecting (1) The epiglottis, which may be completely destroyed ; (2) The parts above the true vocal cords. Necrosis of the cartilages. Perichondritis. Later : laryngeal stenosis.

Symptoms.—Early : hoarseness of the voice, hoarse cough, aphonia. Late : dyspnœa from stenosis.

Treatment.—Iodides and mercury. Tracheotomy for stenosis.

Tuberculous Laryngitis.—Primary, or secondary to phthisis.

ANATOMY.—Tuberculous infiltration and ulceration of arytenoid elevations, aryteno-epiglottidean folds, and epiglottis.

SYMPTOMS.—Great pain—Hoarseness and cough—Dyspnœa and painful dysphagia.

TREATMENT.

General.—Open air in warm climate, and forced feeding. Absolute silence.

Local.—Sucking pastilles of cocaine gr. $\frac{1}{4}$, codeine gr. $\frac{1}{4}$, and morphia gr. $\frac{1}{12}$. Spraying with solutions of the above. Curetting the tuberculous deposits after cocainization, and then rubbing in lactic acid (50 per cent solution).

Diseases of the Larynx, *continued.*

New Growths of the Larynx.

PAPILLOMA.—In young subjects—Generally arises from the vocal cords—Causes hoarseness, dyspnœa, or laryngeal spasm.

TREATMENT.—Removal by intralaryngeal cutting forceps.

EPITHELIOMA.—Patients over forty—Begins as a warty growth —Very soon ulcerates and invades neighbouring structures.

SYMPTOMS.—Cough—Bloodstained expectoration—Hoarseness or aphonia—Pain, and often dysphagia.

EXTRINSIC FORM.—At or above the rima glottidis. Rapidly spreads to the tongue and pharynx. Quickly involves the cervical glands.

TREATMENT: Total laryngectomy. Prognosis is almost hopeless.

INTRINSIC.—Growing from the vocal cords—comparatively benign—Does not affect the glands.

TREATMENT: Thyrotomy, with local removal.

SURGERY OF THE CHEST.

Wounds of the Lungs.

CAUSES.—Contusions—Fractured ribs—Penetrating wounds.

RESULTS.—Contusion or laceration of the lung. Severe pain, shock, and dyspnœa.

HÆMOPTYSIS.—Expectoration of blood-stained mucus or pure blood, with death from asphyxia or syncope.

HÆMOTHORAX.—Rapidly increasing area of dullness, with diminished breath sounds, without evidences of inflammation.

PNEUMOTHORAX, with pulmonary collapse. Severe dyspnœa, with tympanitic chest note and amphoric breathing.

HÆMOPNEUMOTHORAX.—As above, but associated with splashing sounds.

EMPHYSEMA: (1) SURGICAL. Due to the air being forced across the pleural cavity, or sucked into an external wound, into the cellular tissue of the parietes. (2) INTERSTITIAL. Very rare. The air enters the connective tissue of the lung, and may appear in the cellular tissue of the neck.

PLEUROPNEUMONIA, or septic pneumonia.

GANGRENE of the lung.

PLEURISY OR EMPYEMA, especially in cases of penetrating wounds.

TREATMENT OF LUNG WOUNDS.—Absolute rest and recumbency. Avoidance of talking. Firm strapping and bandaging of the chest, unless the dyspnœa is so urgent as to forbid this.

FOR HÆMOPTYSIS.—Ergot, adrenalin, sulphuric acid. These all cause contraction of the vessels and raised blood-pressure. Opium and amyl nitrite act by diminishing the blood-pressure.

FOR HÆMOTHORAX AND PNEUMOTHORAX.—Surgical treatment is seldom necessary. Aspiration may be adopted in the latter case.

For Penetrating Wounds.—Careful treatment of the parietal wound. Examination by X-rays to determine foreign bodies or depressed fractures. Intercostal hæmorrhage must be arrested by enlarging the wound and removing part of the rib if necessary. Pulmonary hæmorrhage is usually beyond treatment if severe. In moderate cases it ceases when syncope occurs.

Direct Suture of the Lung is very dangerous unless it can be done in a special pneumatic chamber in which the air pressure is reduced, the patient's head and the anæsthetist being outside and the surgeon inside the chamber. This prevents pulmonary collapse.

Removal of Foreign Bodies is only to be undertaken: (1) If they are causing definite inflammatory symptoms; (2) If the X rays show that they are in an accessible position. Pleuritic adhesions and pulmonary abscess are common complications, and the adhesions prevent the pleural cavity from being opened.

Chronic Pleural Effusion.

CAUSES.—Tubercle and heart disease. Other infections almost invariably lead to empyema.

PHYSICAL SIGNS.—Side of the chest moves badly. Massive dullness extending from the base upwards. Diminished breath sounds and vocal fremitus. An area of ægophony towards the upper level of the fluid. Displacement of the heart to the opposite side, and of the liver and diaphragm downwards.

SYMPTOMS are those of the primary lung condition—dyspnœa, cough, rapid pulse, etc.

SURGICAL TREATMENT.—Aspiration through the sixth interspace in the mid-axillary line. As much fluid as possible is withdrawn until coughing or bleeding begins.

Empyema.

CAUSES.—Injuries, wounds, pleuropneumonia, tubercle, abdominal suppuration. Extension from neighbouring abscesses, e.g., liver, kidney, subphrenic, etc.

Micro-organisms.—*Diplococcus pneumoniæ* is far the commonest. Tubercle is associated with the very chronic cases. Staphylococci and streptococci are fairly frequent.

SIGNS are the same as those of a serous effusion, but if left to itself, the intercostal spaces begin to bulge, and actual pointing of the abscess occurs usually in one of the upper spaces in front.

SYMPTOMS.—As in the case of an effusion, but a raised or hectic temperature is the rule.

RESULTS.—The lung becomes collapsed, and the visceral pleura then ties it down by becoming thick and contracted. The parietal pleura becomes enormously thick and fibrous. Permanent displacement of the heart and mediastinum, which become dragged over to the diseased side. The opposite lung undergoes some

Empyema, Results, *continued.*

compensatory hypertrophy. The diaphragm and abdominal viscera are displaced upwards. The chest wall falls in somewhat, and the spine is curved with the concavity towards the disease.

PROGNOSIS.

PNEUMOCOCCAL INFECTIONS run a favourable course without becoming chronic.

PYOGENIC AND TUBERCULOUS CASES usually become chronic, and require resection operations to close the cavity.

STREPTOCOCCAL INFECTIONS are usually fatal.

TREATMENT.—If ASPIRATION shows that the fluid is pus, then, except in small effusions with pneumonia :—

DRAINAGE ought to be performed. This is best done at the ninth rib in scapular line, removing a piece of the rib. Place the patient on the diseased side, and rather over on his face. Large but short drainage tube.

IRRIGATION is not often employed, but in chronic cases it may be useful. Use sterile water, with a gentle stream and free outflow.

RESECTION OF RIBS (Estlander's operation).—In chronic cases after the cavity has become permanent. An oblique or flap incision is made over the side. A large piece of all the ribs over the cavity (usually the third to ninth) is removed, or two portions at the anterior and posterior ends are removed so that the middle piece may fall in. As much of the thick pleura as possible is scraped away.

RESECTION OF CHEST WALL (Schede's operation).—A large flap is turned up from the side of the chest, including all the soft parts down to the ribs. The whole of the bony and pleural outer wall of the cavity is removed in one piece. As much of the visceral pleura as possible is dissected off the lung.

Abscess of the Lung.

CAUSES.—Pneumonia—Gangrene—Foreign body—Wounds.

SIGNS.—Are those of local consolidation, with hectic temperature. X rays give a shadow.

TREATMENT.—Free opening through the chest wall, with excision of a piece of rib. If pleural adhesions are firm, the lung can be opened directly after plunging a probe or sinus forceps into the cavity. Drain with a large tube.

If pleural adhesions do not exist, it is best either to stitch the visceral and parietal pleuræ together, or to pack in some gauze and wait for two or three days.

HYDATID OF THE LUNG is treated in the same way.

Wounds of the Heart.—Usually bullet or stab wounds. Death occurs at once from hæmorrhage or syncope in most cases. In those who do not die, the pericardium becomes filled with blood. This causes an increased area of dullness to percussion, with great diminution of the cardiac sounds.

TREATMENT.—A flap including all the thickness of the thoracic wall is turned up over the fourth and fifth ribs and spaces. The pericardium is opened and the wound sutured.

Pericardial Effusion.—Seldom requires surgical treatment. Aspiration should be done in the fifth space, 1½ in. from the sternal margin.

Pericardial Suppuration.—May be acute from pyogenic cocci (including the pneumococcus) or chronic from tuberculous infection.

DRAINAGE is required if the effusion does not yield to other measures. Excise portions of the fourth or fifth costal cartilage.

CHAPTER XXXIII.

DISEASES OF THE BREAST.

Ulcers of the Nipple.

SIMPLE CRACKS AND FISSURES.—Caused by lactation. Result often in abscess in the breast.

ECZEMA.

1. Ordinary acute eczema, recovering under treatment.
2. Chronic eczema, with much scaling, but no destruction of the nipple or induration.
3. Paget's disease. A form of chronic eczema. The nipple becomes destroyed. The deep surface of the eczematous patch is hard and indurated. Carcinoma of the underlying breast follows.

TREATMENT.—First try ointments and lotions for one month if the case appears simple. Excise if no improvement takes place, and microscope. Remove the breast if malignant disease has begun.

SYPHILIS.—(1) Primary chancre, rare in the mother of a syphilitic child. Axillary glands large and hard. (2) Secondary mucous patches. (3) Tertiary gummata and ulcers, very rare.

EPITHELIOMA.—Crater-like ulcer with hard-everted edges. Much destruction of skin and nipple.

SCIRRHUS OR SARCOMA.

INFLAMMATORY DISEASES OF THE BREAST.

Acute Mastitis.

CAUSES.—Infection during lactation, either by the ducts or the lymphatics. Pyæmia. Infective parotitis. New-born infants, generally after injury, e.g. "Breaking the nipple-string." Extension from other structures, e.g., pleura or ribs.

VARIETIES.

SUPRAMAMMARY.—Abscess forms between the skin and breast. Simple incision rapidly cures it.

INTRAMAMMARY.—Abscess forms in the breast substance. At first is limited to one or more lobules. It is partly limited by fibrous radiating septa. The whole breast swells because of retained milk.

Treat by free radiating incision ; break down septa in the cavity so that no " pockets " remain. Counter-opening low down for drain.

Submammary.—Between breast and chest wall. May arise from rib or chest disease. Breast is pushed forward, but not inflamed. Treat by opening below and to the outer side.

Chronic Mastitis.

1. LOBAR.—Remains after lactation, from imperfect involution of one or more lobes. Results from injury or an acute mastitis.
   Symptoms.—Pain of neuralgic character. Worse during menstrual period.
   Treatment.—Rest. Firm bandage. Belladonna.
   Diagnosis from cancer. Flat hand feels no tumour. No craggy edge.
2. LOBULAR or interstitial mastitis.—Women about the menopause. Often in thin and sterile women.
   Pathology: (*a*) Overgrowth of interstitial fibrous tissue; (*b*) Contraction of this fibrous tissue; (*c*) Consequent pressure on ducts and acini; (*d*) Epithelial proliferation; (*e*) Dilatation of acini to form cysts. Cysts are filled with thick, dark, mucoid fluid. There are no intracystic growths.
   Signs.—Nodular feeling in the whole breast. One or more lumps can be seen and felt. The flat hand often feels nothing against the chest wall. Lymph-glands are often a little large and tender. Nipple may be retracted. Often present in both breasts. Pain is inconstant, and is worse at menstruation. Definite cysts form elastic swellings.
   Terminations.—Atrophy—Polycystic or fibrocystic disease—Carcinoma.
   Diagnosis.—From cancer or adenoma.
   Treatment.—Short period of firm pressure, and then excision if improvement has not occurred.

Chronic Abscess.—Often tuberculous, possibly actinomycosis: either originating in the chest wall. Pyogenic form arises during pregnancy or lactation. Hard but elastic swelling. Centre is softer than the margin.

Treatment.—Opening, scraping, and draining.

Cysts of the Breast.

RETENTION CYSTS.—From the dilatation of acini. Often have serous discharge from nipple.

Galactocele.—Filled with altered milk. Arises during or after lactation.

DISTENTION CYSTS.

Involution Cysts.—Occur in interstitial mastitis blocking the ducts. Caused by contracting fibrous tissue.

Irritation Cysts.—From irritation of nipple, causing reflex secretion.

INTERACINOUS CYSTS.

Serous Cysts.—Dilated lymph space. Lined by endothelium. Never gives rise to discharge from nipple.

Hydatid Cysts.

Cysts of the Breast, *continued.*

TUMOUR CYSTS.

Adenoma Duct Papilloma Duct Carcinoma	Cysts have intracystic growths, and often a serous or bloody discharge from the nipple.
Carcinoma Sarcoma	From hæmorrhage or degeneration.

Simple Tumours of the Breast.—Fibro-adenoma—Adenoma—Lipoma and chondroma (very rare)—Duct papilloma.

Adenoma Mammæ.

CONSISTS OF tissue like normal breast-gland tissue.

1. Firm Fibrous Capsule enclosing
2. Epithelial Alveoli.—Generally in a single layer—Without ducts—Often dilated to form cysts—Cysts often contain intracystic growths.
3. Interstitial Fibrous Tissue.

VARIETIES.

Pure Adenoma, or acinous adenoma.—Structure similar to the normal gland.

Fibro-adenoma, or tubular adenoma.—Much the commonest. Fibrous tissue is out of proportion to glandular. Alveoli are drawn out into long canaliculi. A fibrous ingrowth takes place into these canaliculi.

Cysto-adenoma, or serocystic disease.—Alveoli are dilated into cysts. Filled with fibro-papillomatous proliferations. Generally form a large tumour, which may burst through the skin. Often become sarcomatous.

SIGNS.

Age.—From puberty to thirty is the time of their origin.

Cause.—Often follows a blow.

Pain.—Neuralgic, worse at menstruation.

Tumour.—Hard, oval, and elastic. Freely movable in the breast substance. Not fixed to skin, nipple, or chest. Does not produce enlargement of the axillary glands. Often multiple and in both breasts. On removal section is hard, white, foliated, and encapsuled. No juice can be scraped from it.

Terminations.—Remain unaltered. Become carcinomatous or sarcomatous.

TREATMENT.—Removal.

Duct Papilloma.—Small tumour near the surface and near the nipple. An epithelial papilloma growing from the lining of a duct. A cyst about the size of a cherry, containing a warty growth. Serous or bloodstained discharge from the nipple. Growth is not indurated, and is sharply defined. No enlarged glands.

Becomes a duct carcinoma by the epithelium of the papilloma growing down through the wall of the duct.

TREAT by removal of the tumour, and careful microscopy.

Duct Carcinoma.—Similar to the above, but tumour has an indurated base. Lymph-glands enlarge. Less rapid and malignant than ordinary cancer.

TREAT by removal of the breast.

MALIGNANT DISEASE OF THE BREAST.

Carcinoma.

VARIETIES.

SPHEROIDAL CELLS.—Scirrhus—the common type. Encephaloid.
COLUMNAR CELLS.—Duct carcinoma.
SQUAMOUS CELLS.—Epithelioma of nipple.

SCIRRHUS OF THE BREAST.

Etiology.—The proportion of females to males is 100 to 1. More common site for cancer than any other organ except the uterus.

Liability is due to:—(1) Rapid structural changes accompanying puberty, lactation, and menopause. (2) Irritation of lactation. (3) Exposure to injury.

May follow eczema (Paget's disease)—Mastitis—Adenoma.

Age forty to fifty, at about the menopause.

Course.—Lasts from about one to three years. In old women may be a chronic disease and last fifteen years.

Anatomy of scirrhus mammæ.—Hard mass in the breast, looking like white fibrous tissue. Tumour is generally small and the breast contracted. Craggy edge with no capsule. Cut surface is concave, and yields juice on scraping. It cuts with a grating sensation. Is ill-defined from surrounding structures. It infiltrates breast, connective tissue, muscles, and chest wall, the tissues becoming actually replaced by cancer cells.

MICROSCOPICALLY: Columns of spheroidal cells—the cancer cells. Generally three or four rows of cells in each column. Columns divide and branch irregularly. Quantity of fibrous tissue surrounds the column of epithelial cells.

Mode of Extension.

1. DIRECT INVASION OF THE ADJACENT STRUCTURES.—The pectoral fascia, pectoral muscles, skin, chest wall or pleura, may be involved by extension of the primary growth.
2. METASTASIS, OR THE EXTENSION TO DISTANT STRUCTURES.*

PERMEATION OF THE LYMPHATICS, in which the cancer cells grow along the lymph-vessels in direct continuity with the growth, is the mode in which this probably occurs.

PERILYMPHATIC FIBROSIS then takes place on the rupture of those lymph paths which have been first affected, and the cancer cells disappear by this process in the central zone.

THE MICROSCOPICAL GROWING EDGE of the cancer cells can be demonstrated in the lymph vessels of the deep fascia, disposed like a circle round the primary growth, the diameter of the circle enlarging with the age of the case.

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* Sampson Handley.

Scirrhus of the Breast—Mode of Extension, *continued.*

METASTASES occur at any point where the cancer cells have permeated, by a side path or in their direct line of growth, some neighbouring structure, or where perilymphatic fibrosis has failed.

THE LYMPH-GLANDS are naturally the first and most important seat of secondary growth. (*a*) In the axilla the pectoral, apical, and subscapular sets are early involved; (*b*) In the neck the glands in the posterior triangle are infected by extension from the axillary set; (*c*) In the mediastinum the glands may be infected by the lymph-vessels which accompany the perforating branches of the internal mammary, or by extension from the neck.

SKIN NODULES may thus occur in an area which is roughly circular and which has the primary growth as its centre. They never attack the distal parts of the limbs, and are rare, except on the trunk, neck, and head.

BONE METASTASIS, too, occurs with a frequency for individual bones which is in proportion to their proximity to the growth, and chiefly at such points as the deltoid insertion on the humerus, or the great trochanter of the femur, where the lymph-vessels of the deep fascia are continuous with the periosteum. The bones most commonly affected are the sternum, clavicle, humerus, ribs, and vertebræ.

TRANS-CŒLOMIC IMPLANTATION is the process by which the cancer cells, when they have reached the pleura or peritoneum, may fall free in these cavities, and cause growths on the surface of the contained viscera.

Symptoms and Signs.—There are always three stages :—

1. STAGE OF EARLY GROWTH WITH DOUBTFUL CHARACTERS.—Hard nodule in the breast, best felt between the flat hand and the chest. Quite painless, and only noticed when washing. No tenderness. Generally in an outlying lobule of the breast. It is incorporated with the gland substance, but at this stage is fixed to nothing else. It steadily increases in size.

2. STAGE OF ESTABLISHED GROWTH WITH WELL-DEFINED CHARACTERS.

HARD GROWTH, which has a nodular, craggy, ill-defined margin.

FIXED to the suspensory ligaments, joining it to skin, producing dimpling.

To the skin itself later on, when it may ulcerate.

To the nipple, dragging on its ducts and producing retraction.

To the pectoral muscle, fixing the breast so that it cannot be moved up and down the length of the muscle fibres.

To the chest wall, so that the breast cannot be moved across the direction of the muscle fibres.

LYMPH-GLANDS form hard, fixed, nodular mass in the axilla and posterior triangle of neck.

PAIN is marked in proportion to fixation. Is neuralgic and intermittent.

SKIN.—May be affected in one of several ways.

1. Brawny, congested, and fixed to the growth.
2. Ulcerated. Smooth, foul ulcer, whose base is formed by the growth.
3. Nodules of secondary growth over the breast, or at a distance on the skin of any part of the trunk.
4. Cancer *en cuirasse*, the skin forming a layer of cancer-invaded tissue. The "orange skin" or "pig skin" often precedes this, the mouths of the glands being opened and large with excess of secretion.
5. Cancerous lymphangitis.—The lymph-vessels are filled with cancer cells, and lymph exudes from the lymph capillaries, giving an appearance of weeping eczema.

DISCHARGE—from nipple—blood-stained—is only occasionally present.

3. STAGE OF CACHEXIA.—Rapid emaciation. Marked anæmia. Skin becomes loose and inelastic all over the body. Growth is densely fixed or widely ulcerated. Lymph-glands by pressure on the vessels and nerves cause œdema and pain in the arm. The solid brawny œdema may be due to the wide destruction of lymphatics by peri-lymphatic fibrosis. Pleurisy or ascites may occur from metastatic growths. Pain constant and unbearable.

Diagnosis.—In stage 1 the diagnosis is always conjectural until the growth is under the microscope; but until proved innocent every doubtful tumour should be regarded as malignant.

FROM MASTITIS.—In this the flat hand against the breast can define no tumour. The development is after a blow or lactation. The edge is not hard or craggy. Generally tender. There may be retraction of skin and nipple and enlargement of the lymph-glands.

FROM FIBRO-ADENOMA.—In this the tumour floats about in the breast substance. Begins before thirty, generally about twenty. It gives pain of shooting kind, worse at menstruation. Very definite outline.

FROM A CYST.—In this there may be other signs of mastitis. Swelling is elastic or fluctuating. History is a long one.

FROM A CHRONIC ABSCESS OR TUBERCULOUS FOCUS.—In this the history is a long one. Pain or tenderness are early. Centre of swelling is softer than the margins.

FROM SWELLINGS OF THE CHEST WALL, e.g., tubercle or actinomycosis of chest wall, aneurysm, etc. These are obviously fixed to the chest, whilst the breast moves over them.

ENCEPHALOID CARCINOMA.

Differs from scirrhus in the following points :—

Anatomy.—Cancer cells are larger. Columns of cancer cells are thicker. Interstitial fibrous tissue is scanty. Growth is large, soft, and vascular.

Etiology.—Occurs in young women, twenty-five to thirty-five. Often follows lactation or pregnancy.

Course.—Very rapid, ending in about six months.

Signs.—Soft or elastic tumour of some size. Very rapid growth. Often feels hot from its vascularity. Is painful in its early stages. Lymph-glands are affected rapidly. Skin is broken through by a fungating mass. Retraction of skin and nipple are absent.

Diagnosis is never long in doubt.

FROM ACUTE MASTITIS.—In this an abscess forms, upon opening which the swelling and signs subside. All the signs of inflammation. High temperature.

FROM SARCOMA.—In this the tumour is round and circumscribed. Pain is absent. Lymph-glands are not involved early.

OTHER VARIETIES OF CARCINOMA MAMMÆ.

Atrophic Scirrhus.—Women over sixty. Very slow in course, lasting ten to fifteen years. Great excess of fibrous tissue. Very scanty cancer cells. Produces atrophy of breast.

Colloid Carcinoma.—A degeneration of any spheroidal carcinoma. Cancer cells become colloidal.

MICROSCOPICALLY an irregular network of interstitial tissue contains a few degenerate cells and large clear spaces where the colloid takes no stain.

SIGNS.—Enlargement more rapid. Softer character. Does not lessen the rapidity of the disease.

SARCOMA MAMMÆ.

Varieties.

ROUND-CELLED, like lymph tissue. Very rapid. Many metastases, especially in lungs. Lymph-glands seldom involved.

SPINDLE-CELLED—the common. Cells are oat-shaped. Slower in growth. Definitely encapsuled at first.

Etiology.—Any age, without reference to functional changes. Old women and young are often subject to it. No relation to lactation. May follow fibro- or cysto-adenoma.

Signs.

TUMOUR is elastic or semi-fluctuating. Round and well-defined. *Very rapid* growth.

SPREAD.—Skin is first involved. A fungating, bleeding mass protrudes. Deep tissues are invaded later.

METASTASES.—Lymph-glands, especially in spindle variety. Lungs and pleura.

DEGENERATIONS.—Hæmorrhage—Spurious cysts—Myxomatous degeneration.

Treatment of Malignant Disease of Breast.

1. RADICAL REMOVAL always involves :—
   - (*a*) Removal of whole gland with a good margin.
   - (*b*) Removal of wide area of skin over breast.
   - (*c*) Removal of pectoral fascia : better with the sternocostal part of muscle.
   - (*d*) Removal of whole of axillary glands, pectoral, subscapular, and apical groups.

   Occasionally, removal of pectoralis minor in order to clear glands higher ; removal of supraclavicular glands.

   SPECIAL POINTS.

   Cases of doubtful diagnosis : excise tumour, and microscope, without waiting for typical signs.

   Cases where much skin is removed : (*a*) Undercutting edges of skin ; (*b*) Cutting skin flaps ; (*c*) Thiersch's skin-grafting.

2. CASES UNSUITED FOR RADICAL OPERATION.

   Skin conditions : Cancer *en cuirasse*—Widely diffused nodules of cancer—Cancerous lymphangitis.

   Adhesions to chest wall.

   Lymph-glands causing œdema of arm, or forming a mass in posterior triangle of neck.

   Metastases : Pleura, peritoneum, or bones.

   Cachexia, with rapid wasting.

3. METHODS OF TREATMENT FOR INOPERABLE CASES.

   Oophorectomy : Recurrent cases—Women before menopause.

   Large doses of thyroid extract.

   Injections of trypsin for cancer ; Coley's fluid for sarcoma.

   X-ray treatment : For recurrent nodules in the skin. Lessens pain and fixation. Lengthens life.

   * For the brawny œdema of the arm : Insertion of subcutaneous silk threads from the wrist to the chest. These act as capillary drains.

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* Sampson Handley.

CHAPTER XXXIV.

INJURIES OF THE ABDOMEN—PERITONITIS.

CONTUSIONS OF THE ABDOMEN.

Results.

PARIETAL.—Contusion of abdominal wall—Abscess in the abdominal wall—Rupture of abdominal muscle—Subsequent hernia—Rupture of parietal peritoneum—Bleeding from vessel in parietes, from vessel in abdomen.

VISCERAL.

RUPTURE OR CONTUSION OF VISCERA.—Liver—Spleen—Kidney—Bladder—Stomach—Intestine—Pregnant uterus—Ovarian cyst.

PERITONITIS, or PERITONEAL EXTRAVASATION of stomach or bowel contents.—Gas—Blood—Urine.

Symptoms following abdominal contusion.

1. MARKED SHOCK.—Rapid, weak, irregular pulse. Pallor, cold sweat. Slow, shallow, irregular breathing (thoracic). Subnormal temperature. Fainting directly after blow, soon recovering consciousness.

   All these signs ought to improve within twelve hours. If they do not, or if they get worse, visceral injury is indicated.

2. EVIDENCES OF LOCAL INJURY to abdominal parietes.—Swelling and ecchymosis of parietes. Tenderness to the touch and great pain on movement. Absence of these signs, combined with great shock, is evidence of visceral lesion. When parietes are much injured the viscera often escape.

3. SYMPTOMS OF VISCERAL LESIONS.—Signs of shock continue or get worse, except temperature, which often rises. Abdomen is held more and more rigid. Breathing is entirely thoracic.

   Abdomen becomes distended :

   (*a*) WITH BLOOD.—The pulse may be hæmorrhagic—Pallor increases — Restlessness becomes marked — Shifting dullness is found in the flanks.

   (*b*) WITH GAS AND ALIMENTARY CONTENTS.—Tympanites—Loss of liver dullness—Blood-stained vomit—Shifting dullness.

   (*c*) WITH URINE.—Inability to micturate or passage of small quantity of blood and urine—Shifting dullness.

   (*d*) INFLAMMATORY PRODUCTS.—Signs of peritonitis come on from thirty-six to seventy-two hours after injury.

Treatment.—Rest and warmth to combat the shock.

EXPLORATORY OPERATION.

If general and local signs get worse, especially rising pulse-rate, rising temperature, abdominal distention, abdominal rigidity, shifting dullness, open the abdomen and search for the visceral lesion.

PERITONITIS.

Causes.

1. FROM WITHOUT.—Penetrating wounds—Puerperal peritonitis.
2. FROM STOMACH OR INTESTINES.—Injury, ulcer, or perforation.
3. FROM FALLOPIAN TUBES.—Generally gonorrhœal.
4. FROM BLOOD.—Tuberculous—Rheumatic—Pneumococcal.

Bacteriology.*

STAPHYLOCOCCUS ALBUS is commonest and least virulent. Escapes first through injured gut wall in any inflammatory or strangulated condition. Produces phagocytosis and adhesions. Limits spread of peritonitis from other organisms.

STAPHYLOCOCCUS AUREUS.—Rare, and much more virulent.

STREPTOCOCCUS PYOGENES.—Generally from wounds, also from intestine. Very fatal.

BACILLUS COLI.—Almost always present. Generally from intestine. Pure infection is nearly always fatal. Mixed with *S. Albus*, as it generally is, it is not nearly so fatal.

PNEUMOCOCCUS.—Generally a blood-infection associated with pneumonia. Widely diffused. Thin pus. Much fibrin.

TUBERCLE.—Either from intestinal ulceration or blood-infection.

Anatomy.

DILATATION OF VESSELS lying beneath peritoneum. Best seen in the coils of intestine.

EXUDATION of lymph upon peritoneal surface, which becomes sticky. This is seen first in the angles between adjacent coils of intestine.

Exudation of serum and leucocytes and bacteria, to form purulent exudate. This falls into the most dependent parts —loins and pelvis.

SMALL ROUND-CELLED EXUDATION beneath the peritoneum.

ENDOTHELIUM IS SHED in virulent infections. In this case, if recovery occurs, the lymph is organized by the round-celled layer, and forms permanent fibrous adhesions.

If the endothelium is not shed and recovery occurs, the lymph adhesions are absorbed.

ADHESIONS.—First lymph, and later organized granulation tissue (see above), mat together adjacent viscera. The omentum especially adheres to the focus of greatest inflammation.

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* Dudgeon and Sargent.

Peritonitis, *continued.*

Symptoms.

GENERAL.—Profound shock—Drawn face—Small, rapid, wiry pulse—Quick and shallow respirations—Temperature raised, except in worst cases—Vomiting (little force, dark or fæcal)—Hiccough—Constipation.

LOCAL.—Extreme tenderness—Rigidity of the abdomen very marked—Pain local and then diffuse—Breathing purely thoracic—Tympanites—Legs drawn up—Dullness in flanks after two or three days.

Treatment.

GENERAL.—Rest—Hot fomentations—Turpentine enemata.

Mag. sulph. ʒj, alt. horis, if there is no obstruction, until bowels move freely.

Avoid opium, if possible. It obscures the diagnosis, and paralyzes the intestines.

Continuous subcutaneous or rectal saline infusion.

OPERATIVE.

OPEN ABDOMEN: over seat of injury if known; in mid-line if not known.

SEEK CAUSE of peritonitis and deal with it.

SWAB away all exudation.

IRRIGATE with normal saline at 105° F., if adhesions are absent and peritonitis is general.

DRAIN with tubes and wick if pus or fæces are present: (1) Local lesion; (2) Douglas' pouch; (3) Loins.

Local Peritonitis.

CAUSE.—Abdominal injury, or visceral injuries, with infection of low degree of virulence.

VARIETIES.—Abdominal contusion—Subphrenic abscess (*vide infra*)—Leaking gastric ulcer or carcinoma—Cholecystitis, or leaking gall-bladder—Duodenal or other intestinal ulcer—Appendicitis—Pelvic peritonitis—Inflammation of ovarian tumour or of uterine appendages—Inflammation of appendices epiploicæ.

SYMPTOMS.

1. LOCAL PAIN AND TENDERNESS.
   Symptoms referable to organ involved, e.g., dyspepsia, jaundice, uterine discharge.
2. LOCAL SWELLING.—Induration. Dullness to percussion. Increase of visceral symptoms.
3. SIGNS OF LOCAL ABSCESS.—Raised and remittent temperature. Fluctuating or œdematous swelling. Abscess bursting internally or externally.

TREATMENT.—First, rest and hot fomentations. When diagnosis is established, operate—Remove cause.

CHRONIC PERITONITIS.

Causes and Varieties.—(1) Simple irritation, or septic infection of a mild type; (2) Tubercle; (3) Malignant disease.

Simple Chronic Peritonitis is of two different types.

LOCAL FIBROUS PERITONITIS is the result of septic invasion, which has been of a mild character. It may accompany, or remain after, any acute local infection, and is therefore common round the gall-bladder, stomach, appendix, or uterine appendages.

SIMPLE PERITONITIS OF UNKNOWN ORIGIN.—In rare instances general chronic peritonitis arises with effusion and thickening of the peritoneum, but with no clear evidence of its cause. Its association with RHEUMATIC FEVER and BRIGHT'S DISEASE suggests that it is toxic in origin. The peritoneum, especially over the liver and in the great omentum, becomes much thickened and distorted, and there is a marked ascites.

Tuberculous Peritonitis is most commonly found in children and young adults.

ORIGIN.

1. As a diffuse miliary invasion of the peritoneum, the tubercles occurring in the course of the blood-vessels.
2. From the intestine, either appendix, ilium, or cæcum, and other parts more rarely. In this case the lymph tissue of the gut becomes infected, and thence the disease spreads to the peritoneum.
3. From the uterine appendages: this being one of the commonest modes of origin in women.
4. From tuberculous glands in the mesentery.

VARIETIES.—(1) Ascitic; (2) Fibrous; (3) Suppurative, or ulcerous.

THE ASCITIC VARIETY.—This is, perhaps, the commonest, and certainly the most mild variety.

SIGNS.—The usual signs of distention with free fluid are present. There is a little thickening of the peritoneum and mesentery, but adhesions are absent. The fluid is straw-coloured, highly albuminous, and contains many leucocytes.

TREATMENT by: (1) Constitutional and local remedies, among the latter being the application of iodine or mercury ointments; (2) Laparotomy without irrigation or drainage. The beneficent effect is probably due to the outpouring of serum, containing anti-bodies, after the removal of the fluid. It cures about 70 per cent of the cases.

THE FIBROUS VARIETY.

ANATOMY.—There is marked thickening of the mesentery and omentum, the latter often being rolled up as a dense band below the colon. Adhesions form by which the intestines

Tuberculous Peritonitis—The Fibrous Variety, *continued.*

become matted together, especially round the primary focus of disease. Contraction of the mesentery also tends to produce kinking and, together with the adhesions, may cause intestinal obstruction.

THE PHYSICAL SIGNS are those of some free fluid, and also irregular masses can be felt in the abdomen in the situation of the groups of adhesions. The lymph-glands in the mesentery and behind the peritoneum are notably enlarged.

THE TREATMENT is usually only constitutional; the result of operations being as a rule very bad, owing to the ready way in which the diseased intestine is torn, and the impossibility of removing the affected tissues.

THE SUPPURATIVE OR ULCEROUS VARIETY.

ANATOMY.—This is a further development of the last variety. In the midst of the masses of adherent gut and omentum, foci of caseation and suppuration occur. It is common round the tuberculous uterine appendages. The bowel itself is often the seat of advanced tuberculous ulceration. Secondary infection of any such suppurating foci by the intestinal bacteria is common.

PHYSICAL SIGNS are similar to the last case; but localized fluctuating swellings may make their appearance, or break through the parietes, especially at the navel.

TREATMENT is by laparotomy when constitutional treatment has failed. An attempt should be made to remove the primary focus of the disease when this is in the Fallopian tube, appendix, or lymph-glands. There is a grave risk of wounding the bowel and leaving a fæcal fistula.

SYMPTOMS.—These are as insidious and ill-defined as in the case of tuberculous lesions elsewhere. Some hectic is present in acute or late cases. Emaciation, loss of appetite, and indefinite abdominal pain. Intestinal obstruction is common in the adhesive and suppurative types.

Malignant Peritonitis.

CAUSES.

1. Primary malignant disease of the peritoneum. This is an endothelioma, and begins as a rule in the pelvis.
2. Secondary deposits from papilliferous cysts, especially those growing from the hilum of the ovary. These burst through the capsule of the primary growth and become implanted all over the peritoneal surface.
3. Deposits secondary to visceral carcinoma, especially of the stomach, liver, and intestine.

PHYSICAL SIGNS.—Pain, emaciation, and the presence of some tumour, precede the collection of free abdominal fluid.

THE FLUID WITHDRAWN is usually blood-stained, but in the case of the ovarian papilliferous cysts it is thick and mucoid.

CHRONIC PERITONEAL EFFUSION—ASCITES.

CAUSES.

1. Chronic peritonitis, tubercle, new growth, etc.
2. Cardiac and renal disease.
3. Portal obstruction. Cirrhosis of the liver. Malignant disease in the liver or portal glands.

PHYSICAL SIGNS.

BARREL-SHAPED DISTENTION OF THE ABDOMEN, the skin becoming shiny and marked by " striæ."

PERCUSSION shows areas of dullness in the flanks during recumbency, in the hypogastrium in the erect posture, or all over if distention is so great that the mesentery is too short to allow the gut to float against the parietes. In typical cases the areas of dullness become resonant when the patient turns over.

FLUID THRILL is felt on one side of the abdomen when the other is percussed: distinct in proportion to the tension of the fluid.

THE FLUID derived by tapping is clear and almost colourless, and contains little albumen and few blood-cells in the obstructive cases as compared with the inflammatory or malignant.

ANATOMICAL VARIETIES.

1. DIFFUSE.—In this, by far the commoner variety, the fluid occupies the great sac of the peritoneum.
2. LOCALIZED.—(*a*) In the lesser sac. Often arising from pancreatic disease, and giving the signs of pancreatic cyst; (*b*) In the pelvis, especially in women, when it resembles a retroperitoneal or broad ligament cyst; (*c*) In the omentum or mesentery, when it resembles a mesenteric cyst.

TREATMENT.

TAPPING by a trocar and cannula over a dull area. This is done most effectively and with least discomfort to the patient by Southey's capillary tubes, one being introduced into each flank, and allowed to drain for several hours.

EPIPLOPEXY, or Talma's operation, is only suitable for cases arising from portal obstruction. The great omentum is sewn into the parietal wound, and serves to form a new anastomosis between the portal and systemic veins.

SUBPHRENIC ABSCESS.*

Definition.—A localized collection of pus in contact with the under surface of the diaphragm.

Anatomical Division.—The peritoneum is reflected from the upper surface of the liver on to the diaphragm in a cruciform manner at the coronary, falciform, and lateral ligaments. This affords a simple classification :—

1. Intraperitoneal—right anterior, right posterior, left anterior, left posterior.
2. Extraperitoneal—right, left.

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* Barnard, *Brit. Med. Jour.*, Vol. I, 1908.

Subphrenic Abscess, *continued.*

Etiology.—The greatest number occur between twenty and thirty, the sexes being equal. The causes are septic infection from a neighbouring viscus, or very rarely traumatism.

RUPTURED GASTRIC AND DUODENAL ULCERS cause about one-third or more.

APPENDICITIS and HEPATIC DISEASE cause one-sixth each.

The remaining third of the cases are distributed among the following: Parturition, pyæmia, splenic infarct, thoracic disease, gastric cancer, disease of the lumbar vertebræ, cholangitis, suppurating gall-bladder, suppuration of the right kidney, stabs or crushes of the upper abdomen.

Pathology.—The process consists of either: (1) A local peritonitis, which spreads from a neighbouring viscus by contiguity or gravitation, or by the lymph-stream towards the diaphragm; (2) A cellulitis which extends beneath the peritoneum from the liver or cellular tissue of the loins.

THE BACTERIA are the *Bacillus coli* and staphylococci in most cases.

Description of the Varieties.

1. RIGHT ANTERIOR INTRAPERITONEAL ABSCESS.—Over one-third of all cases. Between the right lobe of the liver and the diaphragm, in front of the right coronary and to the right of the falciform ligaments. It spreads down into the loin and below the liver, so that in the majority of cases more than one fossa is involved.

   CAUSES.—Appendicitis, liver and gall-bladder suppuration, gastric and duodenal ulcers, are the commonest, and occur in this order. The liver becomes adherent to the anterior parietes, and therefore does not become displaced.

   SIGNS.—The abscess forms between the right lobe of the liver and diaphragm, pushing the latter up and causing signs of consolidation at the base of the right lung, or in gastric cases it forms a tympanitic swelling in the right hypochondrium, bounded by the falciform ligament mesially and a line between the navel and right costal margin below.

2. RIGHT POSTERIOR INTRAPERITONEAL ABSCESS.—About one-eighth of the cases. Is between the right kidney and the right lobe of the liver behind the right coronary ligament; is nearly always associated with the right anterior form of abscess.

   CAUSES.—Appendicitis in the great majority.

   SIGNS.—An abscess extending below the right costal margin towards the right iliac fossa.

LEFT ANTERIOR INTRAPERITONEAL ABSCESS.—Forms about half the cases. Lies between the diaphragm above, the left lobe of the liver and stomach behind, the adhesions between omentum and parietes below, the spleen to the left, and the falciform ligament to the right. Usually simple.

CAUSES.—Gastric ulcer in the majority.

SIGNS.—An epigastric swelling, bounded by the mid-line and a line from the navel to the left costal margin, in which gas forms a movable area of tympanites.

4. LEFT POSTERIOR INTRAPERITONEAL ABSCESS is the rarest of all varieties (4 per cent). It is contained in the lesser peritoneal sac, having the liver above, the transverse mesocolon below, the stomach in front, and the pancreas behind.

   CAUSES.—Posterior gastric ulcers. Disease of the pancreas and bile-duct.

   SIGNS are very obscure, being those of a swelling behind the stomach having the relations of a pancreatic cyst.

5. RIGHT EXTRAPERITONEAL ABSCESS.—Forms about a quarter of the cases. It forms between the layers of the right coronary ligament above the back of the liver, and pushes the liver down towards the abdomen. The space in which it forms is continuous in front with the space between the layers of the falciform ligament, and behind with the retroperitoneal cellular tissue round the right kidney.

   CAUSES.—Hepatic and biliary suppuration in the majority of cases. Suppuration of the right kidney or head of pancreas, duodenal ulcer, and thoracic suppuration rarely.

   SIGNS.—Consolidation at the right base with marked downward displacement of the liver. The abscess may point in the epigastrium or in the right loin.

6. LEFT EXTRAPERITONEAL ABSCESS.—A rare variety (5 per cent). It forms between the layers of the left coronary ligament.

   CAUSES.—Suppuration of left kidney. Posterior gastric ulcer. Disease of the lumbar vertebræ. Left empyema.

   SIGNS.—Consolidation at the left base, with an abscess pointing in the left loin.

Symptoms.

HISTORY.—Previous symptoms, of dyspepsia, or liver disease, is given in most cases, but the appendix cases usually are of acute origin without previous attacks.

ONSET is sudden in more than half the cases, including nearly all the intraperitoneal varieties. It is insidious in rather less than half, including all the extraperitoneal cases.

PAIN is situated over the site of the abscess, and is acute and stabbing in acute cases, dull and aching in the insidious ones.

Vomiting, hæmatemesis, and melæna are due, when they occur, to the causative gastric or duodenal ulcer.

Diarrhœa follows temporary constipation.

Pyrexia, with all its constitutional signs, complexion, tongue, sweating, etc.

Rigors are exceptional and of very ill omen.

Leucocytosis is constant and well marked.

Subphrenic Abscess, *continued.*

Signs.

ABDOMINAL.—An abdominal EPIGASTRIC OR HYPOGASTRIC SWELLING is found in two-thirds of all cases. In the majority of these it is formed by an intraperitoneal abscess; in the rest by the liver pushed down by an extraperitoneal abscess. The swelling does not move on respiration, because it is fixed by adhesions.

A TYMPANITIC AREA which moves with the patient's posture forms in front of the abscess in many cases.

TENDERNESS AND RIGIDITY are well marked over the hepatic area.

THORACIC.—DULLNESS AT ONE OR BOTH LUNG BASES with diminished breath sounds, due to consolidation or compression of the lung, with or without pleurisy, friction sounds being heard in the latter case.

AMPHORIC BREATHING AND TYMPANITES are found when there is a pyopneumothorax.

THE HEART may be displaced upwards, lateral displacement being much more characteristic of pleurisy.

THE ABSCESS MAY POINT either in the hypochondrium or, more rarely, in the epigastrium or loin.

THE ABSCESS MAY BURST into: (1) The stomach; (2) Pleura; (3) Bronchus; (4) Peritoneum; (5) Intestine; (6) Externally. When this occurs into a viscus or mucous canal it often leads to cure, but into a serous cavity it is rapidly fatal.

EXPLORING SYRINGE (should only be used as the first step of an operation when the patient is anæsthetized, and never through the peritoneal cavity). A deep puncture (3 in.) is made in the tenth, ninth, eighth, or seventh spaces in order from below upwards, first in the scapular line and then in the mid-axillary line. If it has gone through the diaphragm, THE SYRINGE MOVES WITH RESPIRATION.

Diagnosis is made by attention to the history and signs of abdominal disease.

IN PLEURAL EFFUSIONS, especially pyopneumothorax, the history and signs are those of lung disease. The heart is displaced laterally and not upwards. The exploring needle does not move with respiration. The breath sounds are more widely abolished, and the area of amphoric breathing is much larger.

IN HEPATIC TUMOURS AND ABSCESSES the liver moves with respiration, except in malignant disease, and there is much less tenderness or hectic.

IN PNEUMOCOCCAL PERITONITIS WITH PNEUMONIA the peritoneal condition is usually generalized; otherwise it would, *in fact,* constitute a subphrenic abscess.

Treatment.—Free opening and draining directly the diagnosis is made. The operations may be conducted by various routes according to the position of the abscess ; only the first two routes are common.

ANTERIOR ABDOMINAL ROUTE, for anterior intraperitoneal collections, the general peritoneal cavity not being opened. A supplementary drain through the loin is often required, especially in right-sided cases.

POSTERIOR TRANSPLEURAL ROUTE.—The incision follows the track of the exploring syringe. A rib is resected not higher than the seventh, and the diaphragm sewn to the parietes. The abscess is then opened through the diaphragm.

OTHER ROUTES.—Anterior transpleural, lateral transpleural, subpleural, and lumbar openings may be required. The last is often necessary to drain collections of pus in the sub-hepatic pouch.

Prognosis.—About half of all cases die. All cases die which are not operated on. The prospect after posterior operations is rather better than after anterior ones.

CHAPTER XXXV.

APPENDICITIS.*

Anatomy of Appendix.

LENGTH.—Four inches, or anything between three-quarters of an inch and nine inches.

DIAMETER.—Quarter of an inch (unless distended).

POSITION.—Base joins cæcum one inch below the ileocæcal valve—Point marked on surface, middle of a horizontal line between right anterior superior iliac spine and the mid-line.

It is found in the following positions, in the order of frequency named, when inflamed: (1) At the ileo-cæcal angle; (2) Behind the cæcum; (3) In the pelvis; (4) On the outer side of the cæcum; (5) Incarcerated in one of the retroperitoneal fossæ; (6) In front of the cæcum; (7) In the sacs of inguinal, femoral, or umbilical herniæ.

DIRECTION: (1) Upwards and outwards behind the colon; (2) Downwards and inwards over the pelvic brim; (3) Upwards and inwards behind the ileum.

GUIDE TO POSITION.—The muscular tænia coli converge to it.

STRUCTURE.—The following layers are found from within outwards:—

1. Mucous membrane, containing simple mucous glands.
2. Submucous tissue, containing large solitary lymph follicles, each lymph follicle having a basal lymph sinus surrounding it on one side.
3. An ill-developed muscularis mucosæ.
4. A layer of circular muscle fibres.
5. A layer of longitudinal muscle fibres. In one or two places 4 and 5 are deficient, and through this "hiatus muscularis" the submucous and subperitoneal layers are in direct communication, and by this channel bacterial infection often takes place.
6. Subperitoneal connective tissue containing blood- and lymph-vessels.
7. Peritoneum.

ARTERY AND VEIN.—Branches of the ileocolic which run behind the ileum—Branch from left ovarian is probably only a pathological variation.

MESENTERY.—The fold of peritoneum containing the artery in its free border—Attached to the iliac mesentery.

ILEO-APPENDICULAR FOLD AND FOSSA.—A recurrent branch from the artery of the appendix to the ileum lifts up a peritoneal fold and bounds a fossa of this name.

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* C. B. Lockwood, "Appendicitis."

Etiology.

SEX.—Males to Females as four to one.

PREDISPOSING CAUSES.

- Obstruction to the Lumen of the Appendix.—Swelling of mucous membrane—Kinking of a long appendix—Chronic constipation—Fæcal concretions.
- Obstruction to the Circulation.—Owing to vessels lying behind the cæcum. Owing to the artery not running out to the end of the appendix.
- Foreign bodies—very rare.

EXCITING CAUSES.

- Micro-organisms.—Bacillus coli and staphylococci are common. Streptococci, tubercle, and actinomycosis are rare.
- Ulceration or Inflammation of the mucous membrane gives entry to the above.

Pathological Anatomy.

EROSION of mucous membrane by concretion, foreign body, or kinking.

MUCOUS MEMBRANE becomes generally inflamed or ulcerated. Sloughing from pressure, thrombosis, or toxic action of bacteria.

BACTERIAL INVASION occurs through the bases of the mucous glands, or generally through the base of an ulcer. Thence the lymphatic tissue is invaded, and by these the process spreads to the other coats, and eventually to the peritoneum. Bacterial infection of the peritoneum may occur without demonstrable perforation, ulceration, or gangrene.

ARTERY IS OFTEN THROMBOSED.—This may cause gangrene, as there are no collaterals to carry on circulation.

VEINS MAY BECOME THROMBOSED.—Portal pyæmia and hepatic abscess may result.

PERITONEUM.—Plastic inflammation or suppurative inflammation.

WALL OF THE APPENDIX AS A WHOLE undergoes (1) Coagulation necrosis; or (2) Sloughing; or (3) Perforation.

CONCRETIONS occupy the lumen in a large proportion of the worst cases. They are usually formed of a mass of bacteria agglutinated round some fæcal residue. In many cases they bring about ulceration, perforation, or gangrene.

FOREIGN BODIES, e.g., pins, worms, or fruit seeds, are rarely present, and then may be related to the cause of the inflammation.

THE LUMEN, in addition to being occupied by concretions, may undergo one or more of the following changes:—

1. It may be obliterated by destruction of the mucous membrane and the union of the submucous tissue. This rarely affects the entire organ.
2. Stenosis may be produced at one point, as in the last case, or by kinking.
3. Dilatation of the lumen beyond the stenosis by either mucus or pus.

Pathological Anatomy, *continued.*

STRUCTURES IN THE NEIGHBOURHOOD.—(1) Plastic peritonitis; (2) Suppuration limited by adhesions; (3) Diffuse peritonitis.

EXTENSION.—The cæcum, ileum, and peritoneum in iliac fossa are almost always involved. Omentum is generally involved. Pelvis and uterine appendages are often involved. Lower abdominal contents often. Whole of the abdominal contents rarely.

Varieties.

1. Appendicitis without involvement of the peritoneum.
2. Appendicitis with plastic peritonitis.
3. Appendicitis with localized suppurating peritonitis.
4. Appendicitis with generalized peritonitis.
5. Relapsing appendicitis—probably a succession of attacks similar to 1. 2. or 3.

Clinical History.

GROUP 1. APPENDICITIS without peritoneal involvement.

PAIN.—Sudden, sharp, and recurring at intervals; felt in right iliac fossa. Tenderness over this region.

TEMPERATURE.—Little or no rise.

DIGESTION.—Loss of appetite, furred tongue, constipation.

ANATOMY.—Appendix is long, or kinked, and the mucous membrane is often ulcerated and the wall invaded by bacteria.

COURSE.—Mild relapsing attacks.

GROUP 2. PLASTIC PERITONITIS.—Similar to the above, but also:

FEVER.—Temperature rises to 101° or 102° F.

LOCALLY.—A resistant mass is felt over appendix. Most marked after third day.

TENDERNESS at first over region of ileocæcal valve, one third distance between right anterior superior iliac spine and umbilicus: McBurney's point.

COURSE.—Clears up in about three weeks. Very liable to relapse.

ANATOMY.—Local plastic peritonitis matting together cæcum, appendix, ileum, and omentum.

GROUP 3. LOCALIZED SUPPURATING PERITONITIS.

ONSET.—Generally abrupt and severe. Often in the early morning during sleep. Sometimes preceded by malaise and indefinite pain and tenderness.

LOCALLY.—General abdominal distention and tenderness, most marked in right iliac fossa.

Right iliac fossa presents: (1) Muscular resistance and tenderness for first three to five days; (2) Indefinite resistant mass for third to seventh day; (3) Mass becomes dull to percussion. Skin dusky and œdematous.

Rectal examination shows marked tenderness on right side; boggy or fluctuating swelling later.

Right leg drawn up.

TEMPERATURE.—Rises to about 104° F., with occasional rigor. Afterwards either (*a*) Remains high, especially with gangrene; or (*b*) Drops while pulse increases.

RESPIRATION.—Almost entirely thoracic. Increased rate.

PULSE.—Rises to 100–120°. Occasionally remains slow throughout.

ANATOMY.—Appendix is usually perforated or gangrenous, but suppuration may occur in the absence of gangrene, perforation, or ulceration. A collection of stinking pus is surrounded by adhesions.

ABSCESS SPREADS: Down into the pelvis; up behind the colon to right kidney; through the abdominal wall; into upper abdomen.

ABSCESS BURSTS: Into bowel, cæcum, ileum, or rectum; externally; or into peritoneal cavity.

DIGESTIVE ORGANS.—Tongue furred and dry. Constipation generally absolute. Vomiting severe at first and subsiding later. Small quantities of mucus, pus, or undigested food can often be detected in the motions. Rarely blood is passed by the bowel.

MICTURITION is sometimes painful, and this generally indicates that the inflammatory process is adjacent to the bladder.

COURSE.—Acute symptoms last about five days. Then usually subside and give place to those of a local abscess, or merge into (1) General peritonitis; or (2) Portal pyæmia.

GROUP 4. DIFFUSE PERITONITIS.

ONSET and early symptoms as in last group.

LOCAL SIGNS.—Except for the right lower quadrant being most tender, there are no local signs. Signs of general peritonitis rapidly set in: abdominal distention, immobility, rigidity, and tympanites.

TEMPERATURE.—High at first, generally falls, or may be subnormal during the rest of the illness.

PULSE rises to 120, and gets more rapid and smaller.

ANATOMY.—Appendix is perforated or gangrenous. Thin, stinking, seropurulent exudation lies between the coils of intestine, unlimited by any adhesions.

COURSE.—Patient dies in three to six days of general peritonitis.

GROUP 5. RELAPSING APPENDICITIS.

RECURRENT ATTACKS of inflammation or of plastic peritonitis in or round a damaged appendix.

SYMPTOMS or signs are similar to 1 or 2.

RECURRENCE occurs at intervals of months or years. Intervals tend to become shorter. Recurrent attacks may lead to an abscess; seldom or never lead to diffuse peritonitis.

ANATOMY.—Appendix may be (*a*) Kinked, stenosed, dilated, its walls being invaded by bacteria: the peritoneum quite unaffected; or (*b*) Inflamed and bound down in a mass of adhesions.

Diagnosis.

GENERAL SIGNS.—Abrupt onset, with rise of temperature. Constipation and vomiting. Tenderness, pain, and resistance in right iliac fossa are the cardinal signs of appendicitis, of which the local signs of most value are :—

LOCAL SIGNS.—Tenderness and swelling felt per rectum is of great help in diagnosis in a fat or distended subject. Leucocytosis indicates an abscess formation.

THE DIAGNOSIS OF MILD OR RELAPSING CASES has to be made from :—

BILIARY COLIC.—When there is jaundice. Often no rise of pulse or temperature. Tenderness in upper right quadrant.

RENAL COLIC.—When there are pus, cells, or crystals in urine. Affection of micturition. Pain shooting into genitals or leg.

MOVABLE KIDNEY.—Kidney is felt to be mobile. Symptoms referable to micturition.

OVARITIS.—When there is an enlarged, tender, or prolapsed ovary ; often disturbance of menstruation.

THE DIAGNOSIS OF LOCAL SUPPURATIVE PERITONITIS from :—

PELVIC ABSCESS, especially parametritis. Uterus is felt fixed and displaced.

ILIAC OR PSOAS ABSCESS.—Signs of bone disease. Abscess is "cold."

NEW GROWTH OF CÆCUM.—History of constipation and diarrhœa. Abdominal distention of long standing. Mass is hard, well defined, and not tender.

EARLY INTUSSUSCEPTION.—Mass is not specially tender. Alters in size, shape, consistency. Passage of bloody mucus per anum.

COLITIS.—Passage of large quantity of mucus or casts per rectum. Tenderness, if present, is over the whole course of the colon.

INFLAMED UTERINE APPENDAGES—e.g., pyosalpynx or small ovarian cyst—by bimanual vaginal examination.

THE DIAGNOSIS OF ACUTE DIFFUSE PERITONITIS from perforating gastric, duodenal, or intestinal ulcer ; acute pancreatitis ; acute intestinal obstruction ; ruptured gall-bladder ; ruptured extra-uterine gestation ; ruptured pyosalpynx (*vide* Chap. XLIII).

Prognosis is always a matter of doubt.

BAD SIGNS.—Absence of local resistant mass. Persistent high temperature. Temperature low, with rapid pulse. General abdominal distention. Marked constipation with vomiting. Persistent hiccough. Absence of leucocytosis.

Treatment.

GENERAL.—Bed. Hot fomentations. Low diet, e.g., whey or *meat juices.* Enemata : soap-and-water or turpentine.

DRUGS.—Opium only as a preliminary to operation, or if operation is refused. Not when diagnosis is uncertain. Purgatives, especially saline, in milder or recovering cases.

OPERATION, with removal of the appendix, in all cases where the diagnosis is clear.

MAY BE POSTPONED: (1) In mild or relapsing cases so as to wait for a quiescent interval; (2) In mild cases where the diagnosis is doubtful, especially where ovaritis, dysmenorrhœa, or movable kidney exist.

SHOULD BE PERFORMED AT ONCE when general distention and tenderness exist; when there is marked severe onset without recession of the severity of the symptoms; with persistent vomiting, and steadily rising pulse-rate; when there is evidence of suppuration.

OPERATION SHOULD ALWAYS BE PERFORMED when general and local symptoms do not rebate within forty-eight hours; when there is definite evidence of local suppuration.

CHAPTER XXXVI.

DISEASES OF THE STOMACH.*

Examination of the Stomach.

ORDINARY METHODS of inspection, palpation, auscultation.

AUSCULTATORY PERCUSSION enables the outline of the dilated gas-containing stomach to be delimited. Percussion is lightly performed at one spot, while the stethoscope is applied to another over the stomach area.

LAVAGE through a stomach tube.

CAPACITY.—This is normally about two to two and a half pints. Increased by dilatation or atony, and decreased by diffuse carcinoma, or rarely by adhesions.

QUALITATIVE.—Examination of stomach contents for blood, pus, free hydrochloric acid, lactic acid, or sarcinæ.

QUANTITATIVE.—Estimation of free HCl.

DISTENTION OF THE STOMACH is best done through a stomach tube and syringe, but may also be performed by giving a drachm of bicarbonate of soda and then tartaric acid, which blows up the viscus with CO_2.

TEST MEALS.—About six ounces of bread or meat, with some milk or water.

MOTILITY.—The stomach ought to be empty three to four hours afterwards. Recognizable fragments, e.g., currants, fig seeds, can be recovered at long intervals in pyloric obstruction. Salol is only decomposed when it reaches the duodenum, when it can be detected as salicyluric acid in the urine. This ought to occur within two hours of a meal.

ILLUMINATION by a lamp in a flexible tube shows the outline of the stomach when it is distended with fluid.

GASTROSCOPY can be performed through a long straight tube, but only affords a limited view of the parts near the cardiac orifice.

SKIAGRAPHY.—Two ounces of bismuth carbonate are given in bread and milk. This throws a shadow on the fluorescent screen. In normal conditions it demonstrates the distinction between the cardiac reservoir and the actively contracting pyloric antrum.

Foreign Bodies in the Stomach.

VARIETIES.—There are three main categories: (1) Tooth-plates, whistles, etc., swallowed accidentally; (2) Nails, pins, etc., swallowed by lunatics; (3) Hair balls formed of felted masses of hair in women who bite off their hair ends.

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* *Robson* and Moynihan, "Diseases of the Stomach and Their Surgical Treatment."

SYMPTOMS.—These may be absent, or may consist of pain, epigastric tenderness, vomiting, or hæmatemesis. Skiagraphy commonly, and palpation rarely, aid the diagnosis.

TREATMENT.—Removal by gastrotomy.

Congenital Stenosis of the Pylorus consists of a contraction of the pylorus, with dilatation of the rest of the stomach, found soon after birth, or in infants within the first year of life.

PATHOLOGY.—A funnel-shaped circular thickening of the pylorus, half to two inches long, the cavity of which barely admits a probe, The thickening consists in fibrous overgrowth of the submucous tissue and of a mass of hypertrophied muscle. The rest of the stomach is hypertrophied or dilated according to the degree of stenosis and the length of life. Whether the condition is one of developmental error or of spasm followed by hypertrophy of the sphincter, is not known.

It is doubtful whether adult cases exhibiting a similar condition are of the same nature (i.e., congenital) or acquired contraction.

SYMPTOMS AND SIGNS.—Soon after birth or in early infancy.

VOMITING becomes regular and severe. The vomit contains no bile.

GASTRIC DISTENTION soon becomes apparent, so that the whole abdomen may be occupied by the stomach.

PYLORIC TUMOUR can be felt from time to time.

PERISTALTIC WAVE is both seen and felt passing from left to right.

SYMPTOMS.

WASTING is rapid and excessive.

CONSTIPATION is extreme.

TREATMENT.

DIET should be in small measured quantities of peptonized milk or meat juice, and nutrient enemata if possible.

DRUGS.—Minute doses of opium have been given, with good results, possibly by relieving the spasm.

LAVAGE once a day may prevent gastric decomposition.

OPERATION.—Gastro-enterostomy is the best measure, as pyloroplasty is difficult and the condition is likely to relapse.

Injuries of the Stomach.

VARIETIES.—External wounds by stabs or bullets. Internal wounds by injury of foreign bodies swallowed. Internal rupture of the stomach from a contusion (this may affect the mucous coat or the serous coat alone, or all the coats). Spontaneous rupture of an over-distended stomach.

SYMPTOMS.—Pain, vomiting, hæmatemesis will be present in all.

In partial rupture affecting the internal coats, the chief symptoms will be bleeding and collapse.

In cases where all the coats are perforated, the symptoms and signs will be the same as in perforated gastric ulcer.

TREATMENT.—Laparotomy and suture.

ACUTE GASTRIC ULCER.

Varieties.

1. THE SIMPLE EROSION.—Usually multiple. There is very little loss of tissue, but profuse hæmorrhage.
2. THE ACUTE ROUND ULCER, which is the usual type, and is described below.

Causes.—These are quite theoretical.

1. Blood conditions, e.g., anæmia predisposing to auto-digestion.
2. Changes in the gastric juice. Hyperacidity leading to erosion. Possibly certain gastric toxins.
3. Changes in the blood-vessels, leading to embolus and thrombosis, thus accounting for the conical shape of the ulcer.

Anatomy.—Small—less than half an inch in diameter. Any part of the stomach may have them, but the lesser curve or posterior surface are a little more frequently affected than other parts. Often multiple.

CLEAN-CUT, SHARP EDGES.
FUNNEL OR CONE SHAPE.
LITTLE OR NO INDURATION of the base.
READILY HEAL without much contraction.
OPEN THE SMALL VESSELS between the stomach coats.

Etiology.—Young women between puberty and thirty. Associated with chlorosis.

Symptoms.

ACUTE DYSPEPSIA.—Pain is sharp, burning, or cutting. Comes on soon after a meal. There may be a localized epigastric tenderness. The pain is often relieved by vomiting. Generally epigastric. Readily yields to milk diet or starvation.

GENERAL CONDITION.—Fat and anæmic.

Complications.

HÆMATEMESIS may be very profuse. Seldom fatal, as it arises only from the small vessels in the stomach wall.

PERFORATION.

1. GENERAL PERITONITIS.—Especially when the anterior wall is involved.
2. LOCAL PERITONITIS.—Especially when the posterior wall is involved.
3. SUBPHRENIC ABSCESS.

Treatment.—Rest in bed. No food by mouth in extreme cases. Feed by rectum. Later with peptonized milk. Give alkalies and bismuth for slight cases. (For treatment of complications, see below.)

CHRONIC GASTRIC ULCER.

Anatomy.—Often very large—several inches. Usually single. Posterior wall near the pylorus is usual place, 75 per cent being in the pyloric antrum.

EDGES are rounded, indurated and heaped up.

BASE is formed by sclerosed tissues adherent to the stomach.

Produces GREAT CONTRACTION in healing.

In its extension it opens large blood-vessels outside the stomach, e.g., gastroduodenal artery, or splenic. Secondary hæmorrhage results of the typically fatal recurrent type.

Etiology.—Men more often than women. Age thirty to fifty.

Symptoms.

CHRONIC DYSPEPSIA.—Pain: dull, heavy, aching. Occurs half an hour to one hour after food.

PAIN.—Is generally relieved by vomiting. Often in the back as well as epigastrium. Is very slowly relieved by diet and drugs. Is very liable to relapse. Is to some extent localized according to the position of the ulcer. Thus epigastric pain and tenderness relieved by lying on the back points to anterior ulceration. Pain in the back points to posterior ulceration, especially if it is relieved by lying on the face.

GENERAL CONDITION.—Progressive emaciation in proportion to the dyspepsia.

VOMIT.—Excess of free HCl. May be sour and decomposed if gastric dilatation has occurred.

Complications.

PYLORIC STENOSIS.—Dilatation of the stomach.

HOUR-GLASS CONTRACTION.—Dilatation of proximal portion.

PERIGASTRIC ADHESIONS, especially to pancreas and great vessels behind; to parietes in front; to liver and gall-bladder above; to colon below.

THE COMPLICATIONS OF GASTRIC ULCER.

Acute.

1. Perforation.
2. Local peritonitis and abscess.
3. Subphrenic abscess.
4. Abscess of liver, pancreas, spleen.
5. General peritonitis.
6. Hæmatemesis.

Chronic.

7. Local peritonitis and adhesions.
8. Fistula, internal or external.
9. Melæna.
10. Stenosis of the pylorus.
11. Hour-glass stomach.
12. Tetany
13. Pancreatitis.
14. Biliary catarrh and obstruction.
15. Profound anæmia.
16. Carcinoma.

The Complications of Gastric Ulcer, *continued.*

Gastric Hæmorrhage.

CAUSES.

TRAUMA.—Blow or foreign body.

GASTRIC ULCER.—Acute and chronic.

OTHER SURGICAL CONDITIONS, cancer, or aneurysm.

FUNCTIONAL CONDITIONS.—Vicarious menstrual hæmatemesis, post-operative hæmatemesis.

GENERAL DISEASES.—Hepatic cirrhosis, purpura, scurvy, cardiac disease, leukæmia, enteric fever.

HÆMORRHAGE FROM ULCERATION is, apart from traumatism, the only form amenable to direct treatment.

SOURCE OF THE BLEEDING is from capillaries in most acute ulcers, and from arteries in the chronic ulcers and the most severe and fatal forms of bleeding. The splenic, gastro-duodenal, and right gastro-epiploic arteries are those most often eroded.

IN ACUTE ULCER only the small vessels are eroded, and there is no induration to prevent their natural closure.

TREATMENT: (1) Absolute rest. Sucking ice. Ice to the epigastrium. Feeding entirely by the rectum for some days. Morphia for restlessness. Adrenalin chloride, 10 min. of 1–1000 solution, by mouth; (2) Operation is rarely necessary, and only when the above has failed. The stomach must be opened and the bleeding area ligatured. A gastro-enterostomy should be performed also, to keep the organ at rest.

IN CHRONIC ULCER there are usually signs and history of a long-standing dyspepsia. The bleeding may be insidious at first, like that of secondary hæmorrhage. When severe it comes from eroded arteries which cannot contract or retract because they are buried in adhesions.

TREATMENT: (1) Gastro-enterostomy. Gives physiological rest to the stomach, checks the peristaltic movements, and relieves the stomach of its hyperacid and septic contents. (2) Ligature of bleeding vessels on both sides of the ulcer is seldom possible, because of the dense cicatrix in which it is buried.

Perforation of Gastric Ulcer.

CLINICAL VARIETIES.—Acute, leading to diffuse peritonitis. Subacute, causing local peritonitis and abscess. Chronic, causing perigastric adhesions.

ACUTE PERFORATION.

FREQUENCY.—Occurs in 15–20 per cent of cases of gastric ulcer.

SITE OF ULCER.—The anterior wall is perforated in more than two-thirds of the cases. Acute ulcers which perforate are near the cardiac end more often than the pyloric. The reverse holds with chronic ulcers.

SEX AND AGE are those of acute ulcer, viz., in young females.

SIZE of the perforation varies from a minute aperture to one admitting two fingers.

HISTORY OF PRECEDING DYSPEPSIA is usually present, but is absent in about 10 per cent of the cases.

SIGNS OF VISCERAL INJURY.

PAIN, severe, sudden, stabbing, in the epigastrium.

SHOCK and collapse, rapid, severe, and increasing (*vide* Chap. XLIII).

VOMITING is slight and blood-stained, and is exceptional rather than the rule.

RIGIDITY of the upper abdomen, which becomes very hard and tender.

THE LIVER DULLNESS is obscured by free gas.

SHIFTING DULLNESS occurs in the flanks.

SIGNS OF LOCAL PERITONITIS occur in the subacute cases: (1) In the epigastrium, especially round the pylorus; (2) In the region of the right iliac fossa; (3) As a subphrenic abscess.

DIAGNOSIS.—In the absence of a history of dyspepsia a definite diagnosis from other visceral lesions is usually impossible before operation. The inconspicuous character of the vomiting, its blood-stained character when present, and the epigastric location of the tenderness, are the most valuable signs which differentiate it from other perforative lesions (*vide* Chap. XLIII).

TREATMENT.—Open the abdomen in the mid-line above the navel. Examine the anterior gastric surface, beginning at the cardiac end, and then the posterior surface through an opening in the gastrocolic omentum. Close the perforation with a double layer of stitches. If this is impossible, sew omentum over it. Perform a gastro-enterostomy if the ulcer is chronic, or if pyloric stenosis exists. If there is much recent effusion of gastric contents, mop out all the fluid or irrigate thoroughly. If actual peritonitis exists, leave several drains, e.g., in the pelvis, right iliac fossa, and right flank.

PROGNOSIS AFTER OPERATION depends on: (1) Condition of the stomach—the fuller the stomach the worse the outlook; (2) Time between operation and perforation—if less than twelve hours the outlook is good, if more than thirty-six it is almost hopeless in acute cases; (3) Size of the perforation; (4) Presence and severity of the vomiting.

Perigastric Abscess.—Usually due to a subacute rupture of a gastric ulcer.

SITUATION is usually in one of the subphrenic areas, but may rarely be localized in the epigastrium.

EXTERNAL GASTRIC FISTULA is caused by the rupture of such an abscess through the abdominal wall.

INTERNAL GASTRIC FISTULA is caused by perforation into one of the hollow viscera, viz., colon, small intestine, gall-bladder, common bile-duct, or pancreatic duct.

The symptoms, signs, and treatment are given in the section on subphrenic abscess.

The Complications of Gastric Ulcer, *continued.*

Hour-glass Stomach is said to be either congenital or acquired, but the existence of the former is rare and even doubtful.

CAUSES.—Perigastric adhesions resulting from an ulcer. Perforation and adhesion of an anterior ulcer. Cicatrization of a transversely placed median ulcer. Carcinoma, either primary or secondary to an ulcer.

ANATOMY.—The stomach is divided by a transverse constriction into two equal or unequal parts. The constriction is usually about the middle; it may be so narrow that a catheter can scarcely pass its lumen. Great puckering and scarring from ulceration are present, and indicate the causation.

SYMPTOMS are practically those of chronic ulcer associated with those of dilated stomach. But if the constriction is near the cardiac end they may resemble those of œsophageal dilatation.

SPECIAL PHYSICAL SIGNS.

1. Fluid introduced through a tube cannot be all returned.
2. A gush of putrid fluid after washing the stomach clean.
3. Splashing felt after the stomach has been emptied by siphonage.
4. Gurgling sounds heard in the passage of gas and fluids from one cavity to the other.

TREATMENT.

1. Gastroplasty. A division in the long axis of the stomach of the stricture, which is sewn up transversely. Only suitable in the absence of adhesions and induration.
2. Gastro-anastomosis. Making a large anastomotic opening between the two pouches. It is only successful in the absence of pyloric stenosis.
3. Gastro-enterostomy. Uniting the cardiac compartment to the jejunum.
4. Partial gastrectomy. Necessary in malignant and advisable in some simple cases.

Dilatation of the Stomach.

CAUSES.

1. PYLORIC OBSTRUCTION. Cicatrization of a simple ulcer. Perigastritis round the pylorus. Pyloric hypertrophy and spasm. Dragging and kinking by a movable right kidney. Carcinoma. Congenital stenosis.
2. DUODENAL OBSTRUCTION. Cancer. Ulcer. Chronic pancreatitis. Pressure of the superior mesenteric vessels in enteroptosis.
3. ATONIC CONDITIONS associated with dyspepsia and nervous debility.
4. ACUTE DILATATION, probably due to a nerve lesion.

SYMPTOMS.

DYSPEPSIA in varying degrees.

Vomiting at long intervals of large quantities of sour-smelling fluid, in which food particles taken several days previously can be recognized. Increased free HCl in ulcer cases. Lactic acid increased and HCl absent in cancer cases. Sarcinæ are often abundant.

Pain is dull, aching, and accompanied by eructations.

Thirst, Constipation and Scanty Urine arise from the small quantity of fluid passed into the bowel.

Emaciation, Low Temperature, and Asthenia are caused by the bad nutrition.

Tetany, cramps, turning in of the thumb, and convulsions, are seen occasionally in old-standing cases.

SIGNS.

Increased Stomach Area, as shown by inspection and percussion after distention of the stomach with air or gas.

Great Curve Descends below the navel, or even to the pubes.

Succussion Splash is obtainable at all times, except just after gastric lavage.

Visible Peristaltic Wave, from left to right, is seen in chronic pyloric obstruction.

TREATMENT.

Diet.—Small dry meals. Fluid should be taken before meals, or better still, be given by rectum.

Drugs.—Creosote, carbolic acid, and other antiseptics help to check decomposition. Acids or alkalies, according to the reaction of the stomach contents.

Lavage.—Daily washing out the stomach with water or boracic acid will improve all cases with much decomposition, and sometimes cure cases in which there is only partial obstruction.

Gastro-enterostomy is the best radical treatment of all simple obstructive cases. It is useless in all idiopathic or atonic non-obstructive cases. The highest part of the jejunum is united to the posterior wall of the stomach in cases of pyloric disease.

Gastroduodenostomy is performed when the conditions will allow of it, and is the ideal operation.

Pyloroplasty, or the sewing up of a longitudinal incision in the pylorus vertically, is seldom possible because of the thickening round the ulcer.

DUODENAL ULCER.

Duodenal ulceration is similar to that of the stomach, and often occurs at the same time, but there are certain important characteristic differences.

Frequency is about a quarter, or less than that, of gastric ulcer.

Situation.—Almost always in the first part of the duodenum.

Number.—Are often multiple, one being opposite to another.

Gastric Condition.—In about half the cases there is an associated gastric ulcer.

Duodenal Ulcer, *continued.*

AGE.—All ages are liable, and children under ten notably so.

SEX.—Males are much more frequently affected than females: about four to one.

SYMPTOMS are usually similar to those of gastric ulcer, pain, vomiting, and excess of free HCl being the rule. Special characteristics are:

1. Many cases are quite latent and only discovered post mortem.
2. Many cases are quite latent until a severe perforation or bleeding occurs.
3. THE PAIN is dull and aching or burning, felt to the right of the mid-line. It occurs three to four hours after meals, and often appears to be relieved by food (" hunger pain ").
4. BLEEDING is not so frequently observed as in gastric ulcer, because it is not looked for in the fæces. It may occur as hæmatemesis or melæna. Or it may be quite masked in severe or fatal cases, the patient bleeding to death into his own intestine. The gastroduodenal or right gastro-epiploic arteries are those most often eroded.

VARIETIES.—As in gastric ulcer, they are either ACUTE, with a special liability to perforation, or CHRONIC, with liability to hæmorrhage and cicatrization.

COMPLICATIONS.

HÆMORRHAGE.—Is more likely to be fatal and less amenable to medical treatment than in gastric ulcer.

TREATMENT.—Gastro-enterostomy. Rarely it is possible to ligature the involved artery. If the pylorus is not obstructed it is wise to occlude it, so as to prevent food passing through the duodenum.

PERFORATION.—It is more frequent and more fatal than in gastric cases. It is very liable to be mistaken for acute appendicitis, because the exudation gravitates into the right iliac fossa.

In subacute or chronic cases a subphrenic or other ABSCESS, or an internal or external FISTULA may occur.

CICATRICIAL CONTRACTION produces symptoms and signs indistinguishable from those of pyloric obstruction, and the condition must be treated in the same way.

OCCLUSION OF THE BILE-DUCTS, with lithiasis and jaundice.

PANCREATITIS, from occlusion of the pancreatic duct.

CARCINOMA growing in the site of the ulcer.

SIMPLE TUMOURS OF STOMACH.

Varieties.—Adenoma. Myoma. Lymphadenoma. Cysts. These are all very rare, but may cause signs of: (1) A very movable epigastric tumour; (2) Attacks of vomiting caused by the tumour catching in the pyloric aperture.

THE ADENOMA occurs as a polypoid mass with a narrow pedicle. Possibly it is a precursor of malignant disease.

THE MYOMA may grow inside or outside the viscus.

CYSTS grow between the coats of the stomach or between the layers of omentum. They are probably formed by a budding off of an outgrowth from the stomach.

TREATMENT.—Removal, with careful microscopical examination.

SARCOMA.

These form about 5 per cent of all malignant gastric growths.

Varieties.—Lymphosarcoma or spindle-celled.

SYMPTOMS AND SIGNS are the same as in carcinoma, but gastric dilatation is not so common. Also it is more usual for a large movable tumour to be formed than in the case of cancer. The spindle-celled variety may form a polypoid mass capable of local removal.

CARCINOMA OF THE STOMACH.

Etiology.—More common in men than women. Age, forty to seventy, include most of the cases. May occur at thirty, or even younger. Chronic gastric ulcer is sometimes its precursor.

Anatomy.

REGION AFFECTED.—Pylorus in 60 per cent; lesser curve, and cardiac end, about 10 per cent each; other sites together, 15 per cent; general, 5 per cent.

NAKED-EYE APPEARANCE.

1. COMMON TYPE.—Annular growth round the pylorus. Pyloric canal obstructed or obliterated. Growth ends abruptly at the duodenum; extends further along lesser than greater curve. Stomach wall is thickened to half an inch or more. Muscle is hypertrophied in and near the growth. Mucous membrane is often destroyed by ulceration. Peritoneal coat is thick and puckered.
2. SOFT FUNGATING friable mass, with infiltrated base.
3. EXCAVATED ULCER, with everted edges.
4. GENERAL INFILTRATION and contraction of the whole organ —" the leather-bottle stomach."

HISTOLOGY.

1. SPHEROIDAL-CELLED CARCINOMA. — Over 60 per cent. Generally of a scirrhous type. Columns of spheroidal cells invade all coats.
2. COLUMNAR-CELLED CARCINOMA.—Rather less than 40 per cent. Bases of the gastric glands invade the deep tissues. Growth consists of atypical glandular alveoli.

   Both these forms are liable to certain modifications: (*a*) Colloid degeneration; (*b*) Encephaloid; or (*c*) Scirrhous type.
3. SQUAMOUS-CELLED CARCINOMA growing from the œsophagus is excessively rare.

Carcinoma of the Stomach—Anatomy, *continued.*

EXTENSION.

To the Lymph-glands along the lesser curve; in the portal fissure of liver; round the cœliac axis.

To the Peritoneal Surface, and thence by adhesions to liver, pancreas, colon, peritoneal cavity, anterior abdominal wall.

Masses of glands may obstruct the portal vein and thoracic duct.

Submucous Tissue is always invaded much more than the muscular or serous coats, and it is in this tissue that the most advanced edge of the growth is found.

Lesser Curve.—The most marked extension in most cases is along the lesser curve, following the line of the lymph-stream which leaves the stomach with the coronary artery.

Duodenum.—Usually, even in advanced cases, the growth stops abruptly at the pylorus.

Symptoms.

FIRST STAGE.—Dyspepsia, often relieved by food. Pain of a dull, aching character. Steady loss of flesh. Marked anorexia. Hyperæsthetic areas between levels of nipples and navel.

SECOND STAGE.

Tumour.—Small, movable, and at the level of the ninth costa cartilages to right of mid-line, indicates early growth of the pylorus. Indefinite epigastric mass indicates involvement of the body of the stomach, probably its lesser curve.

Vomit.—In cases of pyloric growth. At first only at two- or three-day intervals. Then daily, generally in the morning. Lastly at frequent intervals. Large quantity—one to four pints. Sour smelling—balmy. Mixed with altered blood—coffee grounds.

Vomit in general carcinoma, with contraction of the stomach, comes on soon after food, and is small in quantity.

Gastric Contents.—Absence or diminution of free HCl found in about 85 per cent of cases of gastric cancer. HCl is usually present in cases developing in the site of an ulcer. Presence of lactic and butyric acids—Sarcinæ—Particles of food taken long previously, e.g., seeds or skins—Particles of growth and blood-cells.

Dilatation of the Stomach.—Only present when the pylorus is involved. More than two pints of fluid can be poured into the stomach tube. When inflated with air the great curve descends below the infracostal and often below the umbilical level. Splashing of the gastric contents can be obtained by palpation. Auscultatory percussion shows enlarged gastric area. A peristaltic wave is seen to run from left to right across epigastrium.

Dysphagia.—Only present when the œsophageal orifice is involved.

Blood Examination.—Diminished hæmoglobin. Moderate leucocytosis of mononuclear cells.

THIRD STAGE.—Excessive emaciation. Skin is lemon-yellow or jaundiced—loss of elasticity. Abdomen—hard mass in the epigastrium ; ascites. Œdema of the legs. Severe pain in the back and epigastrium. Mass of growth in the left supraclavicular glands.

Diagnosis ought to be made in the early stages if possible. In the majority of cases, when the second stage is well marked, radical treatment is impossible. Chief diagnostic signs are :—

EARLY.—Insidious dyspepsia occurring for the first time in a patient over fifty. Marked wasting. Diminution of free HCl in stomach contents.

LATER.—Tumour. Signs of dilatation of the stomach.

FROM CHRONIC ULCER.—In this there are : History of long dyspepsia. Wasting is not so marked, and is in proportion to the dyspepsia. Younger patient—thirty to fifty. Bleeding, if it occurs, is in large quantity, and nearly pure blood. Free HCl is increased.

IN SIMPLE CHRONIC DYSPEPSIA, the wasting is not marked. There is no definite alteration in gastric contents.

IN SIMPLE DILATATION of the stomach or with an ulcer, HCl is present. Symptoms and emaciation rapidly improve with lavage.

IN INFLAMMATORY ADHESIONS round the gall-bladder, an indefinite mass may be present. A history of gall-stones or jaundice. Not much wasting. HCl present in stomach.

DOUBTFUL CASES.—All cases in patients over forty-five in whom pain and wasting do not yield to treatment, should be submitted to an exploratory operation.

Treatment.

DIET.—Farinaceous and milk food. Fish, fowl, or finely minced meat. Avoid cellulose, seeds, fibres, skins, etc.

DRUGS.—Acids—HCl.—Nitrohydrochlor. Creosote for fermentation. Opium for pain.

PALLIATIVE.—Lavage once a day, with plain water or Condy.

OPERATIVE.

GASTRO-ENTEROSTOMY.—As palliative measure when pyloric obstruction exists. As preliminary to excision where possible.

GASTRECTOMY.—Removal of growth and lesser curvature, with any glands in portal fissure.

CHAPTER XXXVII.

INTESTINAL OBSTRUCTION.*

Causes.

USUALLY ACUTE.—(1) Strangulation by bands and through apertures, including herniæ, internal and external; (2) Kinking; (3) Volvulus; (4) Intussusception; (5) Obstruction due to foreign bodies.

USUALLY CHRONIC.—(6) Stricture; (7) Tumours growing from the bowel wall; (8) Pressure of tumours outside the bowel; (9) Fæcal accumulation.

Sex and Age.—The male sex are specially prone to herniæ, volvulus; the female sex to pressure by tumours, obstruction by gall-stones, and fæcal accumulation; children to intussusception and congenital stricture; patients over fifty to malignant growths.

General Pathology of Obstruction without Strangulation, as produced by any form of stricture or new growth.

THE BOWEL BELOW THE OBSTRUCTION is empty and contracted. The ballooned rectum below a rectal stricture is the only exception to this.

THE BOWEL ABOVE THE OBSTRUCTION is dilated and hypertrophied in proportion to the chronicity of the case. The mucous membrane is thick, catarrhal, and ulcerated. The ulcers (when the obstruction is in the colon) are specially common just above the obstruction, at the cæcum and in the lower part of the ileum.

THE ULCERS usually cause death by perforative peritonitis, but they may form chronic fæcal abscesses or fistulæ, or very rarely a bimucous fistula, which relieves the obstruction.

SACCULATION OR POUCHING may be marked in either gut, but is commonest in the large. The pouches are often filled with fruit-stones. Gangrene of the gut may rarely occur above the obstruction.

General Pathology of Strangulation, as produced by a hernia or volvulus.

THE INTENSITY OF THE CHANGES and of the symptoms depends upon: (1) The site of strangulation, being much more severe in the small than in the large intestine; (2) The length of gut involved; (3) The tightness of the strangulation, i.e., the degree of vascular occlusion.

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* Treves, "Intestinal Obstruction."

THE GUT ABOVE THE STRANGULATION is red, congested, and distended with gas and fluid. Ulcers or gangrene above the site of strangulation are very rare.

THE GUT BELOW THE STRANGULATION is pale and empty, but very rarely may be the seat of some enteritis.

THE STRANGULATED LOOP becomes congested, œdematous, greatly distended with gas. Later its surface loses its lustre, is covered with sticky exudation, the colour becomes purple and black, and the gut gangrenous.

AT THE LINE OF STRANGULATION ulceration and linear gangrene are common, especially in the proximal loop (for further details, see "Strangulated hernia").

THE CHIEF FACTOR IN STRANGULATION is the vascular occlusion. This produces thrombosis of the vessels, great meteorism from decomposition of the intestinal contents, death of the tissues, with passage through them of bacteria.

Clinical Varieties.—(1) Acute; (2) Chronic; and (3) Chronic ending in acute.

ACUTE OBSTRUCTION.

Causes.—Internal strangulation, kinking, volvulus, foreign bodies, intussusception.

Symptoms.

SHOCK.—Due to an implication of visceral nerves; general prostration; anxious drawn face; great pallor. Subnormal temperature, cold sweats; small, soft, rapid pulse; shallow, quick respiration; vomiting at the time of seizure.

This abdominal shock is common to all forms of "peritonism," and at first there are few or no distinguishing features.

ITS DEGREE is in this case proportioned to: (1) The suddenness; (2) The tightness of strangulation; (3) The nature of gut strangled, small bowel giving more shock than large, and jejunum more than ileum; (4) The amount of gut; (5) The youth of the patient. It passes off or becomes less within twenty-four hours in all except the most acute cases. It is much diminished by morphia.

PAIN.—Of sudden and severe onset. It has four different causes, which act in succession: (1) The actual nipping of the gut; (2) Abnormal peristaltic movements above the obstruction; (3) Distention of the gut; (4) Peritonitis.

IT IS CONSTANT, BUT SUBJECT TO EXACERBATIONS in complete obstruction; it is intermittent in partial obstruction.

IT IS REFERRED TO THE REGION OF THE NAVEL, and gives no indication of the seat of the lesion.

THERE IS A CONSPICUOUS ABSENCE OF TENDERNESS at first; in fact, pressure often relieves the pain, and the patient rolls about in restless agony.

Acute Obstruction—Symptoms—Pain, *continued.*

LOCAL TENDERNESS MAY APPEAR about the third day over the site of the obstruction, and quickly gives place to the general tenderness of peritonitis.

THE PAIN DIMINISHES OR CHANGES ITS CHARACTER when perforation, gangrene, septic intoxication, or peritonitis sets in.

THE PAIN IS INCREASED after aperients, food, enemata, or palpation.

VOMITING.—There are three causes of vomiting: (1) Shock causes sudden vomiting at the onset, especially when the stomach is full; (2) Obstruction of the gut; (3) Peritonitis and distention. The vomiting of obstruction is first bilious and later stercoraceous, becoming so about the fifth day. The tongue is dry and foul.

STERCORACEOUS VOMIT is due to decomposition and bacterial products. It is most conspicuous in small gut obstruction, and occurs early in proportion to the nearness of the obstruction to the stomach. It is caused by the return flow of an axial stream of fluid from the site of obstruction. Its occurrence is delayed by opium.

TRUE FÆCAL VOMITING is excessively rare, and is due to a fistulous communication between the small and large bowel.

CONSTIPATION is absolute, so that neither fæces nor flatus pass in most cases. Is due to reflex nerve paralysis. The lower bowel may empty itself spontaneously or by enemata, but even this is unusual.

SPURIOUS DIARRHŒA, with mucus and blood, which is so frequent in chronic cases, is only seen in acute intussusception among the acute cases.

TYMPANITIC DISTENTION due to meteorism is in proportion to the lowness of the seat of obstruction. When the small bowel only is involved, the central part of the abdomen is most distended, but when the colon is affected, the flanks swell out. It is decreased by strychnine and increased by morphia. Sigmoid volvulus gives the most extreme distention. It is not conspicuous in very acute cases (other than volvulus), but it greatly increases with the onset of peritonitis.

THE ABDOMEN remains flaccid until the onset of peritonitis. Visible peristalsis never occurs in a case which is primarily acute. Its occurrence proves a chronic cause.

TUMOUR is rarely felt in acute cases. (1) Intussusception, (2) Foreign body, (3) Matted intestine, (4) Internal hernia, may give rise to one, but except in the first they are rare.

URINE is diminished or even suppressed by shock. Later the small quantity is due to excessive vomiting. Indicanuria is usual in acute cases when the small gut is involved.

TEMPERATURE is usually subnormal throughout. A slight rise may accompany peritonitis, and an abrupt fall often follows *perforation.*

PERITONITIS supervenes in a large proportion of fatal cases. It is caused by (1) A general transudation of bacteria through the gut, or by (2) Perforation. Its onset is marked by the abdomen becoming tender and rigid, the pulse hard and thready, and where perforation has occurred, gas and free fluid may give the usual signs.

TOXÆMIA may occur with or without peritonitis, and is the last stage of all cases except the ultra-acute ones who die in the stage of shock. Delirium and unconsciousness, dry and cracked tongue, sunken eyes, ashen and livid complexion, restless, with profuse cold sweats, uncountable pulse, sighing respiration.

CHRONIC OBSTRUCTION.

Causes.—A stenosis or obstruction of the gut, which may be due to: (1) Kinking; (2) Adhesions; (3) Shrinking of the mesentery; (4) Some kinds of volvulus; (5) Neoplasms outside or inside the gut; (6) Foreign bodies; (7) Strictures, innocent or malignant; the last being commoner than all the others put together.

Symptoms.

ONSET is gradual, with indefinite " dyspepsia " and ever-increasing constipation.

PAIN is colicky and paroxysmal. Tends to be localized over the seat of obstruction. It is increased by aperients.

VOMITING is irregular and inconspicuous, often absent.

BOWELS.—Constipation is the rule, but it is very important to remember that regular daily actions may occur in spite of marked chronic obstruction. Spurious diarrhœa is common in stricture of the colon or rectum. It is caused by catarrh above the stricture. The motions are then very foul. Bloody mucus is only common when the disease is low down and of a cancerous nature.

SHAPE OF THE MOTIONS.—Occasionally in sigmoid or rectal disease the motions are compressed or tape-like.

ABDOMEN.—Great distention, both by gas and fæces. It is round and barrel-like, or wide and flat. Rumbling and gurgling are heard and felt.

VISIBLE AND PALPABLE PERISTALSIS is caused by: (1) Increased peristaltic efforts above a stricture; and (2) Hypertrophy of the gut. It is therefore well developed in proportion to the chronicity of the case.

CONSTITUTIONAL.—There is much wasting. The complexion becomes muddy and yellow, with yellow conjunctivæ. The temperature rises occasionally, especially after aperients. The constitutional symptoms are proportional to the chronicity of the case rather than to the nature of the obstruction.

Chronic Obstruction, *continued.*

Differences in Chronic Obstruction in the Small and Large Bowels.

IN STENOSIS OF THE SMALL GUT.—General symptoms of indigestion. The pain is influenced by the ingestion of food and the nature of the diet. Meteorism is late. Aperients often relieve. Vomiting is frequent. Tumour is felt in 30 per cent of cases of cancerous stricture. Live three to six months after the onset of symptoms.

IN STENOSIS OF COLON.—Indigestion is absent. Nature of food makes little difference to the pain. Aperients aggravate the symptoms, and may cause collapse, vomiting, perforation, or death. Vomiting is rare until obstruction becomes absolute, and then rarely becomes stercoraceous until the fourteenth day or later. Blood and mucus are common with spurious diarrhœa, especially in cancerous stricture. Ballooning of the rectum and tenesmus may be present when the lesion is low down. Tumour is felt in 40 per cent of cases of cancer. Live three to nine months after onset, but may live two years after colostomy.

CHRONIC OBSTRUCTION BECOMING ACUTE.

Causes.—May occur in any variety of chronic obstruction, when it may be brought on by: (1) Occlusion of a stricture by solid fæces; (2) Kinking of the gut; (3) Peritonitis at and above the stricture; (4) Purgatives; (5) Morphia.

DIFFERENCES FROM A PRIMARY ACUTE ATTACK.—The shock, pain, and suddenness of onset are much less. Visible peristalsis only found if a chronic obstruction has preceded the acute.

Diagnosis of Intestinal Obstruction from:—

1. ACUTE ABDOMINAL DISEASES OF OTHER KINDS: Biliary colic—Renal colic—Lead Colic—Ruptured gastric ulcer—Ruptured gut—Perforated appendix—Acute pancreatitis—Twisted ovarian cyst—Extra-uterine gestation—Ruptured pyosalpynx—Torsion of the testis.

   The diagnosis is made clear generally after the lapse of a few hours by the development of some special symptoms (see Chap. XLIII).

2. ACUTE PERITONITIS from a perforated appendix, stomach, or gall-bladder. In this a rigor commonly occurs with the onset. An early rise of temperature is usual though not invariable. Tenderness is extreme, local at first, more diffused afterwards. The pain in peritonitis tends to subside sooner than in obstruction. The vomiting is less copious, less persistent, and less likely to be stercoraceous. Constipation is not so absolute. The abdomen is hard and rigid. Meteorism is early and diffuse. The patient lies still, with the knees up, as compared with the restless movements of one with obstruction. The pulse is very hard, of high tension. Leucocytosis appears in the majority of cases.

3. TUBERCULOUS PERITONITIS is especially likely to be mistaken for intussusception or for chronic obstruction. There is usually some fever and local tenderness.
4. CHOLERA.—Cases of acute obstruction with profound shock following diarrhœa, or cases of acute intussusception, have been mistaken for this.
5. DYSENTERY, MEMBRANOUS COLITIS, AND ACID INTOXICATION have been confused with intussusception.
6. THROMBOSIS OF THE MESENTERIC VESSELS.—An acute abdominal seizure occurs, with pain, vomiting, and collapse. Blood-stained diarrhœa may occur. The mesenteric veins or one of the arteries may be affected. In the latter case infarction of the gut supplied arises. The diagnosis is seldom made before operation.

Treatment.

1. NON-OPERATIVE.
   - Morphia.—Relieves the pain and shock. Lessens peristaltic contractions, and therefore allays the vomiting. It causes a freer flow of urine by diminishing the nerve inhibition. It prevents the rapid development of an intussusception.
     - Dangers.—It obscures diagnosis and tends to paralyze the bowel. It should, if possible, be withheld until the diagnosis is clear, and then given in doses of ¼ gr. in adults and ⅛ gr. in children.
   - Enemata.—A simple soap enema, one pint in bulk, given as early as possible. Generally it is returned unchanged. It should not be repeated. If it washes out the lower bowel it makes operative measures easier; it also aids in subsequent feeding by rectum.
   - Purgatives should under no circumstances be given if actual obstruction exists or is suspected. They will increase pain, shock, vomiting, and strangulation.
   - Feeding.—All feeding by mouth is absolutely stopped. Thirst is best allayed by half a pint of water at blood heat given per rectum every four hours. A long drink of water (preferably hot) may be occasionally allowed. It causes vomiting, and thus washes out the stomach.
   - Washing out the Stomach often gives temporary relief from vomiting. It is specially indicated before the administration of an anæsthetic.
2. OPERATIVE.
   - Every case Should be Submitted to Operation when complete obstruction is proved or probable. The prospects of success depend largely upon the Early Period at which it is undertaken.
   - The Anæsthetic.—The stomach should first be washed out, to minimize the danger of inhalation of vomited matter. As little anæsthetic as possible is given. Often local anæsthesia or Spinal Anæsthesia will be desirable.

Treatment, *continued.*

THE INCISION should be median below the umbilicus. The only cases in which other incisions are justifiable are: (*a*) When the cause is known to be cancer below the sigmoid flexure, when an incision as for inguinal colostomy will be made; (*b*) When there is a possibility of the case being one of acute appendicitis.

IN DESPERATE CASES.—A mere enterostomy of the nearest available coil of distended gut is sometimes all that can be done. It may relieve the urgent symptoms and allow a more radical operation later.

THE SEARCH FOR THE SEAT OF OBSTRUCTION.—The cæcum is first examined. If it is distended, the large bowel is at fault; if collapsed, then the small gut is the seat of obstruction. The pelvis and left iliac fossa are next examined. The large bowel can easily be palpated in its whole course. The small bowel usually has to be brought out on to the abdomen.

PUNCTURE OF THE GUT.—When great distention exists and affects the colon, and especially the sigmoid flexure, a trocar is thrust in and the gas allowed to escape. A single stitch serves to close this afterwards. It is of little use to puncture distended small bowel, because this relieves only the coil punctured.

ENTEROSTOMY should always be performed after the cause of obstruction has been removed, if great distention, especially of the small bowel, exists. Tie in a Paul's tube.

THE TREATMENT OF GANGRENE OF THE GUT.—In desperate cases it is only possible to bring the dead parts outside and perform an enterostomy above the disease. But if the patient's condition allows it, an immediate enterectomy is to be performed.

Variety I.—Internal Strangulation.

Internal strangulation under bands and through apertures.

Causes.—The strangulating agents are of six kinds.

1. PERITONEAL BANDS.—These result from any form of local peritonitis, especially appendicitis, also from tuberculous disease in the intestine or glands. They become stretched and rolled into cords by intestinal movements. They are often attached to the region of the cæcum, the mesentery, the uterus and ovaries, or the umbilicus. They act in two ways: (*a*) As a short band forming an arch, beneath which the gut becomes snared; and (*b*) A long band which forms loops in which the gut is knotted.

2. CORDS FORMED BY OMENTAL ADHESIONS.—These are similar to the above, a strand of omentum becoming attached to the cæcal region, pelvis, or hernial orifice. Commoner on the left side.

3. MECKEL'S DIVERTICULUM is the remains of the vitello-intestinal duct. It is present in about 2 per cent of bodies. It is about 3 in. to 6 in. long, attached to the ileum 2 ft. to 3 ft. from the cæcum. It may have a lumen of the same size as the gut, or its lumen is obliterated, and it forms a vitelline ligament. Its end may be free or attached to the umbilicus, mesentery, or any other spot. It causes obstruction: (*a*) As a short band under which the gut is caught; (*b*) As a long cord which snares the gut; (*c*) As a free diverticulum with a knobbed end which knots itself round a loop of gut; (*d*) By producing kinking of the gut over it or by its traction; (*e*) By producing an intussusception or volvulus; (*f*) By a stricture formed at its junction with the gut.
4. FALSE DIVERTICULA are hernial protrusions of the mucous membrane through the other walls of the gut. They occur: (1) Towards the mesentery of the small intestine; (2) Into the appendices epiploicæ of the colon. The latter may perforate or become inflamed, and so give rise to obstructing bands.
5. ADHESIONS OF NORMAL STRUCTURES: (*a*) The appendix may be adherent by its tip to the mesentery, ileum, cæcum, bladder, or parietes; (*b*) The Fallopian tube; (*c*) A part of the mesentery may form a tight band by the fixation of a portion of the gut; (*d*) Appendices epiploicæ; (*e*) Ovarian pedicle.
6. SLITS AND APERTURES.—These may be: (*a*) In the mesentery as the result of traumatism or congenital defect; (*b*) In the omentum; (*c*) In a membranous adhesion.

The Mechanism of Strangulation.

It is nearly always the small gut which is caught, and generally the lower part of the ileum.. When once caught, (*a*) the congestion produced by pressure on the veins, (*b*) gaseous distention, and (*c*) a twisting of the loop to form a volvulus, bring about strangulation.

Clinical Characters.

ETIOLOGY.—Usually young adults, because these are the patients so liable to the different forms of peritonitis, hernia, or injury. In over 60 per cent there is a history of one or other of these conditions previously.

ONSET is sudden and abrupt, in the majority of cases, and sometimes is determined by a straining movement, large meal, or purgative.

PAIN is early, severe, and continuous. Vomiting is constant, copious, and severe, and becomes stercoraceous about fifth day.

COLLAPSE, with profound prostration, is well marked.

ABDOMEN shows nothing characteristic. Very rarely a local dullness or an indefinite mass is caused by the engorged coils above the obstruction.

Internal Strangulation, *continued.*

Course of the case ends fatally in about one week, the extremes being eight hours and twenty days. They seldom live more than three days after the onset of stercoraceous vomiting.

Prognosis.—Apart from operation it is always fatal. Death is early in proportion to the tightness of strangulation, the height of the bowel involved, the length of bowel caught.

Death is due to septic absorption, collapse, peritonitis, or perforation, in this order.

Special Points in Operative Treatment.—Bands and adhesions must be carefully divided between ligatures; diverticula removed, and the stumps sutured.

Internal Herniæ.*

DIAPHRAGMATIC HERNIA.—In about half the cases the orifice in the diaphragm is congenital, and in the other half it is traumatic. The stomach, colon, and small intestine are involved, in this order. The symptoms may be acute or chronic. Diagnosis is practically impossible until the abdomen is opened.

TREATMENT will generally involve opening the pleural cavity to close the rent, but no successful case has yet occurred.

DUODENO-JEJUNAL FOSSA.—There are at least nine different varieties of these fossæ, the commonest formed by a peritoneal fold passing from the terminal part of the duodenum towards the left, and making a pouch looking upwards or downwards. The others are, chiefly, the paraduodenal, formed by the fold of peritoneum lifted by the inferior mesenteric vein, and those running under the root of the mesentery or into the transverse colon.

THE LEFT DUODENO-JEJUNAL HERNIA is eight times as common as the other. It is probably into the paraduodenal fossa. It extends to the left behind the descending colon, and has the inferior mesenteric vein and a branch of the left colic artery running in front of its neck.

THE RIGHT DUODENAL-JEJUNAL HERNIA extends behind the peritoneum towards the right behind the ascending colon. It enters a fossa in the root of the mesentery, and has the superior mesenteric vessels in front of its neck.

THE SYMPTOMS vary from those of dyspepsia and uneasiness, to chronic or acute intestinal obstruction. In the latter case the course is acute.

TREATMENT is difficult because of the large vessels in front of the neck of the sac. No successful case is recorded.

FORAMEN OF WINSLOW.—Very rare form, and only possible when an abnormal mesocolon exists. Usually involves the large intestine, but sometimes a large part of the small gut may be involved. The gut may further break its way through the gastrohepatic omentum or through the transverse colon, and then become strangulated. Over the neck of the sac at the foramen of Winslow run the portal vein, hepatic artery, and bile-duct. The symptoms are usually those of chronic obstruction with epigastric distention, followed by acute obstruction.

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* Moynihan.

TREATMENT has always been unavailing because the neck of the sac cannot be divided.

INTERSIGMOID HERNIA.—The intersigmoid fossa is in the root of the sigmoid mesocolon, looks downward and to the left, is bounded by the sigmoid artery. The symptoms are those of internal strangulation.

PERICÆCAL HERNIA.—In the cæcal region: (1) Two fossæ may lie in front of the cæcum, determined by branches of the ileocolic artery. These are sometimes called the ileocolic fossæ; (2) One fossa may lie behind the cæcum and extend up as far as the kidney; (3) One fossa lies behind the mesentery of the appendix; (4) The ileo-appendicular fossa is bounded by the ileum above, appendix below, and the ileo-appendicular fold in front; this is often termed the ileocæcal fossa. Actual cases have occurred, chiefly in (2) and (4). The symptoms may be latent, or may resemble those of internal strangulation by other means.

II.—Kinking.

Causes.

A loop of bowel may be KINKED OVER A BAND. Adhesions or volvulus of the loop usually complicate this rare condition.

A loop of bowel may be KINKED BY THE TRACTION of a band or diverticulum.

ADHESIONS OF THE GUT to form V- or N-shaped loops. This is usually an after-effect of an inflamed or strangulated hernia, the symptoms coming on weeks or months after the hernia.

The CONTRACTION OF ADHESIONS may compress the gut. This occurs round the colon, especially at the hepatic flexure, from gall-bladder inflammation.

The MATTING TOGETHER of several coils of intestine may affect the small or large bowel. In the former it results from local peritonitis, and produces subacute obstruction; in the latter it results from chronic constipation and ulceration, and produces chronic obstruction.

Clinical Signs.—These cases are so rare that they do not form a definite clinical group. They may conform to any type of obstruction.

III.—Volvulus.

Definition.—A kinking of a loop of gut, by either: (1) Rotating round its own mesenteric axis; or (2) Falling across the pedicle of another loop of gut.

Locality.—Three regions may be affected, in the following order of frequency: (1) The sigmoid loop, i.e., the pelvic colon; (2) The small intestine; (3) The cæcum and ascending colon.

Volvulus, *continued.*

The Sigmoid Volvulus.—The loop and its mesentery are usually of abnormal length; the mesenteric attachment is narrow. The peritoneum of the mesocolon is dense, contracted, and often adherent. Chronic constipation, with distention of the loop, is the prominent predisposing cause. The proximal limb usually falls in front of the distal limb. The twist may be from half to three complete turns. As long as distention exists the volvulus cannot be reduced, or re-forms after reduction. In fatal cases the sigmoid becomes enormously distended, until it presses on the liver and diaphragm, the latter being raised up to the level of the third or fourth rib. The loop is intensely congested and hæmorrhagic. Its outer coats are often ruptured. Gangrene is frequent. The mesocolon is engorged. Peritonitis is of constant occurrence. The gut above is distended and sometimes perforated. Sometimes the sigmoid loop is intertwined with a loop of small intestine. The patients in the common variety are usually elderly females.

The Enteric Volvulus.—A loop of small gut has an abnormally long mesentery, or one whose attached border has been puckered by adhesion, e.g., by a caseating gland. A gall-stone in the bowel may bring about the twist. The coil, which may be from one to five feet in length, is enormously swollen, congested, or gangrenous. It is often filled with blood, but death occurs before perforation. Rarely two coils may be intertwined. Enteric volvulus occurs in patients under forty, and sometimes in children.

Cæcal and Colic Volvulus.—The rarest variety. The gut may be twisted: (1) Upon its own axis; (2) At right angles to its own axis, i.e., kinked; (3) Twisted as a loop round its mesocolic axis. The cæcum or ascending colon are the parts affected, and they are always the subject of congenital malposition or possessed of a mesocolon. Rarely an ascending colic volvulus may be intertwined with a loop of small bowel.

Clinical Aspect of the Sigmoid Volvulus.

ETIOLOGY.—Females are four times as commonly affected as males; usually the age is between forty and sixty. There is always a history of chronic constipation.

PAIN is early and severe, but is often intermittent or subject to exacerbations.

VOMITING is late and insignificant, rarely stercoraceous.

SIGNS OF PERITONITIS are early and constant.

ABDOMINAL CONDITION.—Rapid and enormous meteorism occurs from the distention of the sigmoid loop, which may be seen through the parietes and extends up to the diaphragm. Tenderness and rigidity are early because of peritonitis.

RESPIRATION is embarrassed by the rapid abdominal distention.

The other forms of volvulus are too rare to permit of a clinical grouping. They usually cause acute, but sometimes intermittent *or chronic,* obstruction.

Course and Prognosis are rapid (about one week) and fatal.

Death is due to asphyxia from pressure on the diaphragm, septic absorption, or peritonitis.

Special Points in Operative Treatment.—In sigmoid and colic volvulus the loop must be brought to the surface, evacuated through a small incision, untwisted, stitched to the parietes to prevent re-formation of the twist, and a colostomy completed.

IV.—Intussusception.

Definition.—A prolapse of one part of the gut into an immediately adjoining part. It causes one-third of all cases of intestinal obstruction.

THREE LAYERS OF BOWEL are concentrically placed one inside the other. Peritoneum is in contact with peritoneum and mucous membrane with mucous membrane.

The outer layer is the SHEATH or INTUSSUSCIPIENS.

The middle layer is the RETURNING LAYER, and has its coats reversed, i.e., the mucous membrane is outside towards the sheath, and its peritoneum inwards towards the inner layer.

The inner layer is the ENTERING LAYER.

The inner and middle layers, whose apposed peritoneal coats usually adhere together, are known collectively as the INTUSSUSCEPTUM.

THE NECK is the junction of the sheath and returning layer where these grasp the inner layer.

THE APEX is the junction of the middle and inner layers.

Varieties.

1. ENTERIC (include 30 per cent of all cases).—Composed entirely of small intestine. Usually only about 6 in. in length. The jejunum is four times as liable as the ileum.
2. COLIC or rectal include 18 per cent of all cases. Composed entirely of large intestine. Most common in the region of the sigmoid flexure. Usually quite short.
3. ILEOCÆCAL includes 44 per cent of all cases. The ileocæcal valve travels down the colon, dragging with it both ileum and cæcum. It is of large size, the apex formed by the ileocæcal valve often reaching the anus.
4. ILEOCOLIC.—Includes 8 per cent of all cases. The ileum prolapses through the ileocæcal valve, which, together with the cæcum, remains in its place. Secondary ileocæcal invagination usually occurs later.

Anomalous or Rare Forms.

1. Intussusception of the dying. Usually in the small gut, of small size, often multiple, may be retrograde, probably occurs during rigor mortis.
2. Retrograde intussusception is very rare, apart from those of the dying. Usually of short length, and in the colon.

Intussusception—Anomalous or Rare Forms, *continued.*

3. Double, or even triple intussusceptions, are rare, and are caused by one intussusception becoming bodily prolapsed into the gut below, once, or even twice.
4. The iliaca-ileo-colic is an intussusception of the ileum in which the apex formed by ileum reaches the ileocæcal valve and passes through it or pushes it in front of it.

The Mode of Growth is the same in all varieties except the ileocolic.

Growth Takes Place at the Expense of the Sheath, i.e., more and more of the sheath becomes drawn in.

The Apex Remains the Same Throughout, e.g., in the common ileocæcal variety, whether small or large, it is formed by the ileocæcal valve.

This is due to the fact that the inner and middle layers, whose junction forms the apex, have their peritoneal coats in apposition, and these adhere rather than glide over one another. But the outer and middle coats have mucous surfaces next to each other, and these readily glide, so that the intussusceptum slips down inside the intussuscipiens.

Pathological Anatomy.

THE MESENTERY becomes dragged into the intussusceptum, between the inner and middle layers. (1) It is stretched by the traction of the growing intussusception. (2) It is constricted at the neck, where its bulk is largest. (3) Its tissues swell by exudation of inflammatory products.

THE BOWEL ABOVE is congested and dilated; only rarely in chronic cases is it ulcerated.

THE SHEATH OR INTUSSUSCIPIENS is little changed in acute cases; in chronic cases it is affected by local peritonitis, or it may be ulcerated or gangrenous, especially opposite the apex of the intussusceptum.

THE INTUSSUSCEPTUM.

1. It is curved by the traction of the mesentery so that its apex is directed against the wall of the sheath.
2. Œdema is most marked at the apex, which forms a globular tumour, and at the convex border.
3. Catarrh and desquamation of the mucous membrane occur, and account for the mucus and shreds in the stools.
4. Both its layers become engorged with blood, which exudes into the lumen.
5. Gangrene is common in acute cases, and is most marked at the neck; in chronic cases it is most marked at the apex.
6. Separation and passage of the intussusceptum may affect the whole mass, which is passed as a complete double tube a few inches to a yard or more in length, or the tube may have unfolded itself, and so present only a single layer, or it may merely come away piecemeal, the latter being more common in chronic cases.

OBSTRUCTION is caused by (1) The orifice at the apex being dragged upon by the mesentery, so as to be slit-like and apposed to the sheath; (2) The curved kinking of the intussusceptum by the mesentery; (3) The narrowing of the lumen by the pressure of œdema, hæmorrhage, and inflammatory exudation; (4) The blocking of the narrow lumen by some food débris.

STRANGULATION is caused by a compression of the vessels in the mesentery, by the tissues at the neck, by swelling of the layers of the intussusceptum, and by traction and torsion.

IRREDUCIBILITY is caused by (1) Adhesions between the inner and middle layers, these occurring as a rule in chronic cases and exceptionally in acute cases; (2) Swelling of the intussusceptum, especially at its apex; (3) Bending or twisting of the intussusceptum; (4) A polyp or growth at the apex.

Clinical Aspects.

ETIOLOGY.

Sex and Age.—Preponderance of males, especially in early life and in chronic cases. In acute cases 50 per cent occur before ten years and 25 per cent before one year. In chronic cases 50 per cent occur between twenty and forty.

Predisposing Causes.—Diarrhœa, worms, masses of undigested food, polypus, new growth in the gut wall, Meckel's diverticulum, invaginated appendix (the last three very rare).

Exciting Causes.—The irregular peristaltic contraction of the intestine induced by any of the above, either spasm or (rarely) paralysis causing a piece of gut to be swallowed, as it were, by the gut below.

Influence of the Ileocæcal Valve.—This acts like the sphincter ani, and tenesmus draws the cæcum over the ileum.

Cause of Growth of an Intussusception.—When once the condition is begun the peristaltic contractions of the sheath tend to drive down the intussusceptum.

CLINICAL VARIETIES.—These are: (1) Acute, which end fatally within one week, and form 50 per cent of all cases; (2) Subacute, which last up to a month, and form 30 per cent; (3) Chronic, which live more than a month and form 20 per cent.

Symptoms of Acute Intussusception.

ONSET is sudden, with pain or tenesmus.

PAIN is early. Severe at first, and may be less later when the intussusception becomes fixed. It is characteristically intermittent, the paroxysms coming on regularly and lasting a definite time. When a tumour appears the pain is localized to that region. On the whole the pain is less acute than in any other form of acute obstruction.

VOMITING is frequent at the onset, but irregular and inconstant during the course of the disease. It affords more relief than in other cases of obstruction. It varies inversely with the diarrhœa. It rarely becomes stercoraceous. It is most marked in enteric forms.

Symptoms of Acute Intussusception, *continued.*

BOWELS.—Marked constipation is exceptional, and comes on after diarrhœa.

Diarrhœa arises early from catarrh and excessive peristalsis; it also occurs late, and is specially offensive when the intussusceptum is sloughing.

Blood and Mucus occur conspicuously in 80 per cent of the acute cases, especially in children.

TENESMUS is often very prominent. It is an early symptom, and occurs in proportion to the proximity of the lesion to the anus. Patulence of the anal sphincter may occur.

SHOCK, THIRST, DIMINUTION OF URINE, are less marked than in other forms of acute obstruction.

RIGIDITY of the Abdomen and Meteorism are slight and come on late.

TENDERNESS over a localized area may indicate the position of the lesion.

ABDOMINAL TUMOUR occurs in over 50 per cent of all cases. It is most frequent in ileocæcal and colic varieties. More distinct in children. It is sausage-shaped and curved. Most often seen over the descending or transverse colon. It gradually moves in the direction of the path of the colon; it may be made to retrace its path by forced enemata. It hardens during the attacks of pain and may disappear between.

RECTAL TUMOUR.—A mass which feels like a soft os uteri may be felt per anum, or may protrude from the bowel. This occurs in about one-third of the cases, especially in children, in whom it may appear in the second day of the attack. It only occurs in colic and ileocæcal varieties. From three to eight inches may protrude, and the ileocæcal valve and appendix orifice may be recognized at the apex.

The Course of the Case.—The acuteness is determined by: (1) The site of the lesion—the higher being the more acute; (2) The youth of the patient. Ultra-acute cases in infants under one year may die within twelve hours. Eighty per cent die within eight days.

Mortality among all cases is 70 per cent. Those that recover spontaneously are mostly the subacute cases.

Methods of Spontaneous Recovery are (1) Natural reduction, especially after opium; (2) Spontaneous elimination of the intussusceptum. This occurs most frequently in adults. It affects about 30 per cent of the cases, and of these perhaps only 10 per cent recover; the remainder die from perforation, ulceration, or hæmorrhage of the bowel. The time of separation varies from three days to six months, occurring in most cases at the end of the first month. It occurs more frequently in the enteric than in the other varieties.

Chronic Intussusception.—Usually of the ileocæcal variety, and in adults rather than children. May last from a month to a year, the course being very irregular, and vomiting, pain, and diarrhœa all being variable in occurrence. Blood-stained motions with mucus occur in about half the cases. Visible peristalsis is well marked. An abdominal tumour is felt in about half the cases, and felt per rectum in about one-third. The mortality is very high—over 90 per cent.

Treatment.—Morphia is especially valuable as combating shock and preventing the rapid increase of the invagination.

REDUCTION BY ENEMATA.—About one to two pints of hot normal saline is run into the rectum by a funnel and tube, the former held three feet above the patient. An anæsthetic is given during this procedure, and, if unsuccessful, operation is proceeded with. It is only to be employed within the first twelve hours of onset, and is unsatisfactory because: (1) There is danger of rupture of the bowel; (2) The case may soon recur, owing probably to only partial reduction having taken place, the œdematous apex remaining unreduced.

OPERATION IN REDUCIBLE CASES.—Median incision. The tumour is brought into the wound. The apex is squeezed in an ascending direction through the sheath. It is useless and dangerous to drag on the entering layer at the neck. The greatest difficulty is in the final reduction of the œdematous apex. The appendix may be brought through the parietes and cut off to form a stoma through which feeding can take place. This also anchors the ileum and prevents recurrence. Or a simple stitch may be placed in the last part of the mesentery, fixing it to the parietes.

OPERATION IN IRREDUCIBLE CASES.

BARKER'S OPERATION.—If the sheath is healthy, first tuck a little more intussusceptum into the sheath, then sew the two together by continuous stitch; open the sheath by a longitudinal opening and withdraw the intussusceptum; cut it off transversely, sewing together the two layers in the stump.

TOTAL RESECTION of the intussusception is only indicated when the sheath is gangrenous. The formation of an enterostomy is nearly always fatal in children.

V.—Obstruction due to Foreign Bodies.

1. **Swallowed Foreign Bodies** of an indigestible nature.

(*a*) Metal and other hard substances swallowed by accident, e.g., tooth-plates, or by design by lunatics or showmen. These usually pass per anum, but they may become lodged anywhere, especially in the cæcum, and cause obstruction. More commonly they perforate the peritoneum, the parietes, or some other viscus, e.g., the bladder.

Obstruction due to Foreign Bodies—Swallowed Foreign Bodies, *contd.*

(*b*) Sharp foreign bodies, e.g., pins or nails. These may accumulate in the stomach or duodenum, or perforate the alimentary canal and wander to distant parts without causing symptoms.

(*c*) Accumulated masses of débris. Hair, thread, fibres, fruit-stones or skins. Hair-balls may be found in lunatics and hysterical women who chew their own hair.

2. **Gall-Stones.**—Gall-stones which cause obstruction always enter the gut by ulcerating their way from the gall-bladder into the duodenum. Less often they enter the hepatic flexure of the colon in the same way, but then very rarely cause obstruction. They often become added to in the gut by deposits of magnesia or carbonate of lime. Impaction occurs in the lower ileum, or the duodenum or jejunum. Patients are generally women over fifty.

3. **Enteroliths,** or stones formed in the intestine. These may be: (*a*) Phosphates, which are probably deposited from catarrhal secretions; (*b*) Avenoliths formed by calcareous salts and oatmeal débris; (*c*) Mineral deposits from medicines, e.g., magnesia, bismuth, salol, etc. Actual obstruction by any of them is very rare, and then usually occurs from impaction in the lower ileum.

Clinical Aspects of Intestinal Obstruction due to Gall-Stones.

SEX AND AGE.—Three-quarters of the patients are females, and three-quarters are over fifty.

HISTORY.—Previous attacks of biliary colic or of local peritonitis round gall-bladder.

ONSET is abrupt. PAIN is severe but intermittent.

VOMITING is early, incessant, copious, and often stercoraceous.

CONSTIPATION AND COLLAPSE are in proportion to the acuteness of the attack.

The majority of cases are acute, but subacute and chronic cases occur.

UNUSUAL CASES OF GALL-STONE OBSTRUCTION.—Rarely after an acute onset, sudden relief may be caused by the stone passing the ileocæcal valve. A few weeks or months later the stone may pass the anus. Or there may be several successive attacks of subacute obstruction at varying intervals. Or the case may present all the characters of chronic obstruction, such as that due to stricture.

THE CLINICAL ASPECT OF OBSTRUCTION DUE TO OTHER FOREIGN BODIES.—This is similar to that due to gall-stones, except that in the case of enteroliths the case is decidedly chronic, and often ends by the natural passage of the concretion.

Course and Prognosis.—65 per cent die. Death or recovery occurs about the end of a week.

Treatment.—The abdomen is opened in the mid-line. The bowel is incised longitudinally rather above the foreign body. The latter is removed, and the wound sewn up.

VI.—Stricture of the Intestine.

1. **Cicatricial Stricture.**—Produced by the healing of an ulcer.

A. INFLAMMATORY ULCERATION.—(*a*) Duodenal ulcer. Very rarely causes stricture, and then the symptoms are rather those of dilated stomach. (*b*) Tuberculous ulcer. The strictures may be multiple, they are most often in the last part of the ileum or at the ileocæcal valve. (*c*) Typhoid ulcers very rarely cause stricture. (*d*) Syphilitic ulcers: very rare cause of stricture, except in the rectum. The ileo-cæcal valve may be affected. (*e*) Dysenteric ulcers cause stricture in the rectum, sigmoid flexure, descending colon, splenic and hepatic flexures. Taking the above all together, the large gut is affected by stricture six times as often as the small.

B. AFTER HERNIA.—Follows ulceration or limited gangrene of the gut due to strangulation. The symptoms follow from one month to several years after reduction. The ileum is the part generally affected.

C. AFTER INJURY.—This may be: (*a*) Surgical operations, e.g., anastomosis, when mechanical methods like Murphy's buttons are a more frequent cause than simple suture; (*b*) After a blow on the abdomen which has produced a partial laceration of the gut. Here adhesions generally form and help in the production of obstruction; (*c*) Foreign bodies, e.g., gall-stones or tooth-plates.

2. **Cancerous Stricture.**—Carcinoma is always cylindrical-celled. It may produce obstruction in various ways: (*a*) Most commonly by a limited annular band, like a narrow tape, tied round the gut; (*b*) By a fungating mass filling up the lumen of the gut; (*c*) By producing a kinking; (*d*) Very rarely by causing an intussusception.

Dense and extreme contraction of the growth, fungation towards the lumen, ulceration at or above the growth, and colloid degeneration, may be found in the affected part. Adhesions to, or invasion of, surrounding structures may occur, but are much later and more exceptional than in malignant growths elsewhere. Metastatic growths occur in the liver, glands, and lungs, but these are late and often quite absent, even in advanced cases. Carcinoma may occur at any point, but is commonest at the sigmoid flexure, the descending colon, the splenic and hepatic flexures, and the ileocæcal valve.

3. **Congenital Stricture.**—These are all very rare compared with 1 and 2. They occur most frequently in the ileum, a few feet from the ileocæcal valve, where they may be due to an over-obliteration of the vitelline duct; then the duodenum or jejunum. They may amount to an absolute discontinuity of the gut, narrowing to a fibrous cord, a membranous diaphragm, or any degree of stricture. In the colon a very few cases have been recorded. Symptoms of pyloric obstruction in the duodenal cases, or of intestinal obstruction in the others, occur in severe cases, and are fatal a few days after birth. They may remain latent for a few years or throughout life.

Stricture of the Intestine, *continued.*

Locality of the Non-Congenital Strictures.—The colon, and especially the sigmoid flexure, is much more commonly affected than the small gut, sixty per cent of all cases of stricture being in the sigmoid.

In the small intestine, 60 per cent are caused by cicatrization—38 per cent by ulcers, 15 per cent by hernia, 7 per cent by trauma, 40 per cent by cancer.

In the large intestine (excluding the rectum), 65 per cent are caused by cancer, 35 per cent by cicatrization.

The Mechanical Conditions and Effects of a Stricture.—A stricture will not cause absolute obstruction as long as the contents are fluid. Hence fatal small-gut strictures are much tighter than those in the large gut. Actual obstruction may be caused by: (1) Blocking by fæcal material or fruit débris; (2) The production of kinking or volvulus.

Treatment of stricture of the intestine.

FEEDING should be by bland diet which leaves but little residue. Fruit, nuts, vegetables, etc., especially to be avoided.

APERIENTS must be used with caution. Salines are generally of most use, and in cases where the small gut is involved. They must, of course, never be used when obstruction is absolute.

OPERATION.

FOR SLOW MALIGNANT STRICTURE an enteroplasty or resection is indicated.

FOR LONG STRICTURE BURIED IN ADHESIONS a short circuit will have to be done.

FOR MALIGNANT STRICTURES.—EXCISION is the ideal. It ought to be preceded by a short-circuiting operation or colostomy if the patient's condition is very bad, or if great distention exists above the growth.

In inoperable growths the choice lies between A SHORT-CIRCUITING OPERATION, which should always be done, if possible, and an inguinal COLOSTOMY or cæcostomy, the lumbar colostomy having been almost abandoned.

VII.—Other New Growths of the Intestine.

These are all rare and seldom cause obstruction.

1. POLYPI may be adenomata, fibromata, fibromyomata, or submucous lipomata—rarely cystic of congenital origin. They occur most commonly in the rectum (80 per cent), and next in the ileum or colon. They are commonly multiple, and when small cause no symptoms. When they cause intestinal obstruction, this is similar to that caused by stricture. They may separate at their pedicles and pass per anum.
2. LYMPHADENOMA AND LYMPHOSARCOMA occur as submucous growths.

VIII.—Obstruction by Pressure outside the Bowel.

Causes.

UTERINE.—Large retroverted uterus, especially with: Fibroids or pregnancy—Extra-uterine gestation—Pelvic cellulitis—Cancer of the uterus.

OVARIAN.—Growths of any kind, but especially solid or malignant.

VESICAL.—New growths, notably cancer; also large calculus or prostatic new growth.

MESENTERIC.—New growths, especially cysts, subperitoneal tumours, or hydatid cysts.

RENAL.—New growths. Rarely by the pedicle of a movable kidney.

PANCREATIC.—Cancer, or rarely cysts.

SPLENIC.—Large and movable spleens.

HEPATIC.—New growths from the liver or bile-ducts, or glands in the portal fissure.

Regions Affected.—The rectum, 60 per cent; descending and pelvic colon, 12 per cent; lower ileum, 10 per cent; duodenum, 7 per cent; other parts, 11 per cent.

Clinical Characters.—These are of all varieties, viz., acute, intermittent, chronic, or chronic ending in acute obstruction.

IX.—Fæcal Accumulation.

Causes.

1. DEFECTIVE EXPULSIVE POWER, due to some nerve condition or general debility, possibly the over-use of aperients.
2. INHIBITION OF DEFÆCATION. — Resulting from piles, fissure, or any pain in the pelvis, inflamed appendix or ovary; careless habits.
3. A LARGE QUANTITY OF INDIGESTIBLE FOOD and small quantity of liquids.
4. SOME ANATOMICAL ABNORMALITY.—Dilatation and prolapse of the cæcum, sigmoid or transverse colon. Exaggerated sacculi. Adhesions round various parts of the colon, especially near the flexures. Thickening, contraction, and kinking by parts of the mesocolon.

Physical Characters and results on the bowel.—It occurs in the rectum, sigmoid flexure, or cæcum, in this order, or in the whole large gut. The accumulations are of two kinds: isolated masses of stony hardness from which nearly all moisture has been absorbed, and more ordinary fæcal masses. They are often covered with mucus or shreds of mucous membrane. The colon becomes dilated, elongated, and hypertrophied; it may measure 6 in. in diameter; its folds are exaggerated, and its sacculi unduly prominent. Stercoral ulcers are frequent, and may lead to perforative peritonitis.

Fæcal Accumulation, *continued.*

Clinical Characters.

COMMONER IN WOMEN THAN MEN. After middle age and in hypochondriasis.

CONSTIPATION is habitual, but attacks of spurious or catarrhal diarrhœa sometimes occur. Bowels may not act for weeks, or even months.

DIGESTIVE DISTURBANCES.—Poor appetite, offensive breath, foul tongue, eructations, flatulence, and dyspepsia.

MENTAL CHANGES.—Headache, vertigo, apathy, languor, hypochondriasis, or even insanity.

SKIN is dull, dark, greasy, and of an unpleasant odour; the conjunctivæ dull and discoloured.

TEMPERATURE rises occasionally, especially after aperients.

ABDOMINAL DISTENTION may become marked, and produce dyspnœa and palpitation. Visible peristalsis is unusual.

PRESSURE SIGNS.—Sciatica, menstrual disturbances, cold feet, or œdema of the legs.

OBSTRUCTIVE ATTACKS, with great pain and vomiting, occur from time to time, and may prove fatal.

TUMOUR is felt in the course of the large gut, and is formed by masses of fæces. Most common situation is the cæcum, then the sigmoid. Very rarely it is doughy. It is often tender from stercoral ulceration.

DEATH is rare, as the cases yield to treatment; but acute obstruction, perforation, septicæmia, or cardiac failure may cause it.

Treatment.

1. DRUGS, especially saline aperients. The more powerful cathartics usually do more harm than good.
2. LARGE DAILY ENEMATA.—(*a*) Soap and water; (*b*) Olive oil, given a pint over night and retained till next day, when it is followed by a simple enema.
3. SYSTEMATIC EXERCISE and massage where practical.
4. ILEO-SIGMOIDOSTOMY* when all other remedies have failed. The ileum is completely divided and implanted into the sigmoid flexure. The large gut may be excised down to the splenic flexure.

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* Arbuthnot Lane, "Operative Treatment of Chronic Constipation."

CHAPTER XXXVIII.

HERNIA.

Definition.—Protrusion of an internal viscus through an abnormal opening in the parietes.

Signs of Abdominal Hernia.

1. SWELLING, generally situated in the inguinal, femoral, or umbilical regions, or in site of a scar.
2. AN IMPULSE is imparted to this swelling on straining. The impulse is of an expansile character. The swelling is not only pushed out; it also gets larger.
3. REPLACEMENT can be effected into the abdomen of the contents of the hernia (unless irreducible), and this is often sudden and gurgling. When the contents of the hernia reappear, the swelling re-forms from above downwards.

NOTE.—Signs 2 and 3, which prove the connection between the contents of a swelling and the interior of the abdomen, cannot be obtained if the hernia is irreducible, and in this case the diagnosis is a matter of conjecture.

Causes.

CONGENITAL.—Patency of the funicular process. Late descent of the testis. Weakness of the parietes, with large rings. Length of the mesentery. Congenital apertures in the linea alba. Malformation of the umbilicus or of the diaphragm.

ACQUIRED.—Violent exercise (by sudden increase in intra-abdominal pressure). Wearing tight belt, girth, or stays. Anything which causes frequent straining: Bronchitis—Large prostate—Constipation—Phimosis. Pregnancy and parturition. Fat or tumours in the abdomen. Slipping down of the mesenteric attachment. Senile atrophy of abdominal muscles.

Structure of Hernia.

SAC.—Peritoneum pulled down through aperture. In early cases the sac can be pushed back. In established cases the sac is fixed outside the parietes.

Inflammation produces: (1) Thickening of the sac; (3) Adhesion of its contents; (3) Loculation—hour-glass contraction or hydrocele of the sac.

COVERINGS are formed by all the structures which originally cover the hernial orifice. Tend to become matted together at the hernial orifice.

Structure of Hernia, *continued.*

CONTENTS.

Small Intestine.—Enterocele.

Omentum.—Epiplocele, especially likely to become irreducible. May develop serous cysts.

Loose Bodies.—From detached tags of omentum or appendices epiploicæ.

Colon or Cæcum.—Rare, but found in children.

Bladder.—Either the fundus or a lateral saccule. In very large hernias.

Any viscus except the pancreas may descend into a hernial sac in various conditions of enteroptosis.

INGUINAL HERNIA.

Varieties.

1. OBLIQUE OR INDIRECT.—Acquired—Congenital (vaginal or funicular)—Infantile—Interstitial.
2. DIRECT (always acquired).—Internal or External.

Orifice of Protrusion from the abdomen.

The Oblique all go through the internal abdominal ring. External to the deep epigastric artery.

Direct Hernias leave the abdomen internal to the deep epigastric artery, i.e., in Hesselbach's triangle, between the outer border of the rectus and the epigastric artery.

External Direct hernias leaves the abdomen between the epigastric artery and the obliterated hypogastric.

Internal Direct hernia, between the hypogastric artery and outer border of the rectus.

Coverings of an inguinal hernia. From without inwards :—

OBLIQUE VARIETIES.

1. Skin, fasciæ.
2. Intercolumnar fascia from the external oblique.
3. Cremasteric layer from the internal oblique.
4. Infundibuliform fascia from the transversalis fascia.
5. Subserous fatty tissue.
6. Peritoneum forming the sac.

EXTERNAL DIRECT hernia.—As above, except the transversalis fascia instead of the infundibuliform.

INTERNAL DIRECT hernia is covered by transversalis fascia and conjoined tendon, instead of the infundibuliform fascia and cremasteric layer.

INFANTILE HERNIA has two additional peritoneal coverings next to the sac.

Degree of an inguinal hernia.

COMPLETE.—Emerge through the external abdominal ring and descend into the scrotum or labium.

INCOMPLETE or bubonocele.—Lie between the internal and *external* rings under the aponeurosis of the external oblique.

Acquired Oblique Inguinal Hernia.—Sac formed by abdominal peritoneum. Develops slowly and is often incomplete. In early stages is above and distinct from the testis. In late stages it lies in front of the testis. Structures of the cord are spread out over the sac.

Congenital Inguinal Hernia.—Sac formed by funicular process. May arise in infancy. May arise in adolescence (in this case it forms rapidly and soon becomes complete). Commoner on the right side. Very liable to strangulation. Structures of the cord are intimately adherent to the sac.

IN FUNICULAR VARIETY.—Sac lies above and distinct from tunica vaginalis and testis.

IN VAGINAL VARIETY.—Sac is continuous with the tunica vaginalis. Testis is enveloped by the hernia.

Infantile Inguinal Hernia consists of an infantile hydrocele and an inguinal hernia. The funicular process is obliterated at the internal ring, but patent thence downwards, and opens into the tunica vaginalis. The hernial sac is (1) Pushed down behind the hydrocele sac, or (2) Invaginated into it. Thus two layers of peritoneum, with fluid between them, cover the sac.

Direct Inguinal Hernia is always acquired. Generally in late life. Caused by a laxity and weakness of the internal oblique and transversalis muscles. Comes straight through the abdominal wall. Internal and external orifices in the abdominal wall are large and opposite one another. Epigastric artery runs on its outer side. Often remains small and incomplete.

Much more difficult to retain by truss or cure by operation than the oblique varieties. Because of: (1) Large size of the orifice; (2) Laxity of the abdominal muscles; (3) Directness of the descent.

The cord is distinct from and to the outer side of the sac.

Interstitial Hernia.—A congenital oblique hernia in which the sac has an extra diverticulum. This lies :—

1. Between the peritoneum and muscles in front of the bladder, or in the iliac fossa—intraparietal variety; or
2. Between the external and internal oblique muscles parallel to the outer half of Poupart's ligament—interparietal variety; or
3. Between the muscles and skin, along the front of Poupart's ligament: generally associated with retained testis—extraparietal variety.

Especially liable to give rise to difficulty in reduction by taxis.

Signs of an uncomplicated inguinal hernia :—

ROUNDED SWELLING in the groin above Poupart's ligament or extending into the scrotum or labium.

IMPULSE on coughing or straining. The swelling comes down further and also increases in size—expansile impulse.

Inguinal Hernia—Signs, *continued.*

CONTENTS CAN BE REDUCED into the abdomen: Upwards and outwards in indirect varieties. Directly backwards in direct varieties. Reappears from above downwards. Gurgling generally accompanies reduction.

RELATIONS.—Poupart's ligament and groin fold lie below. Pubic spine is below and on outer side in complete varieties. Inguinal canal is occupied by the sac and its contents.

Diagnosis of incomplete inguinal hernia from:

RETAINED TESTIS.—Testicular sensation on pressure. Absence of testis from the scrotum. Impulse is only pushing, not expansile. Upper limit of the swelling can be felt.

SWELLINGS OF THE CORD—Hydrocele—Lipoma—Hæmatocele. —Upper limit can often be felt. Reduction is impossible or very gradual. No expansile impulse. Traction on testis pulls the swelling down.

FEMORAL HERNIA.—In this the swelling appears first below Poupart's ligament. The inguinal canal has only the cord in it. The neck may be felt to be deep to Poupart's ligament.

ABSCESS in the groin, especially a chronic psoas or iliac. A large intra-abdominal swelling exists. Signs of primary disease. Outline is indefinite. Fluctuation exists.

GLANDS IN THE GROIN.—Non-reducible. No expansile impulse. Tender, with indefinite outline. Primary source of infection present. Solid feeling.

Diagnosis of Complete Inguinal Hernia from:

FEMORAL HERNIA.—Sac lies below and outside pubic spine. Reduction occurs downwards and then upwards. Other signs mentioned above.

VARICOCELE.—Feels like a bag of worms. Does not generally extend into inguinal canal. Disappears on lying down. Reappears from below upwards.

CONGENITAL HYDROCELE.—Generally in infants. Elastic or fluctuating. Translucent. Reduction is very gradual. No impulse as a rule.

Diagnosis of Inguinal Hernia when Irreducible or Inflamed.

WHEN INCOMPLETE:

From GLANDULAR SWELLINGS may be impossible. Primary source of infection. Gradual development. Normal inguinal canal.

ACUTE ABSCESS, especially if it contains gas. Limits are outside the inguinal canal. Primary cause, e.g., parametritis, can be found.

WHEN COMPLETE:

From SOLID ENLARGEMENTS OF THE TESTIS.—The cord is thick in tubercle and new growth. Enlargement is slow and *gradual.* Feels solid and heavy. No impulse.

Treatment of uncomplicated Inguinal Hernia.

TRUSSES.

IN INFANTS.—May produce a cure in about one year. Should be rubber-covered. Should never be removed without supporting the inguinal region.

IN ADULTS.—Can only support the rupture. Will never cure the hernia.

MEASUREMENT.—Round pelvis, below crest, to meet over symphysis = size in inches.

SPRING.—Must press backwards and a little upwards. Requires renewal two or three times yearly.

ADJUSTMENT.—Only apply after hernial contents have been reduced. Pad should lie over the inguinal canal and not on the bones. Leg strap is necessary to keep hernia from coming under it. May generally be left off at night.

COMMON MODIFICATIONS.—(1) Rat-tail truss: pad is produced into a tongue process attached to leg-strap; (2) Forked tongue truss: pad has two processes attaching it to leg-piece and to the cross-strap. Both useful for large scrotal hernias in which external ring is very large.

RADICAL OPERATION.

INDICATIONS.—Healthy adults. Active occupation.

CONTRA-INDICATIONS.—Infants under one year, in which a truss often cures. Senility. Lax atonic muscles. Enteroptosis. Existence of chronic bronchitis, enlarged prostate, any cause of straining.

DOUBTFUL CASES.—Direct inguinal hernias—because the muscles are lax or torn. Large hernias of old standing. Large irreducible hernias in old men.

METHOD.—Bassini's operation, or modification of this.

Incision half an inch above inner end of Poupart's ligament (a curved incision turning down a flap of skin is better).

Slit up the external oblique from the external to the internal ring half an inch above Poupart's ligament.

Define the sac and separate it from the structures of the cord. Separate adhesions and remove redundant omentum. Empty the sac after opening it. Tie the neck of the sac (after transfixing it) flush with the peritoneum. Cut off the sac.

SEW THE ARCHED FIBRES OF THE INTERNAL OBLIQUE AND TRANSVERSALIS TO POUPART'S LIGAMENT UNDERNEATH THE SPERMATIC CORD. (This is as well, or better, done over the cord, leaving the latter between transversalis fascia and the conjoined tendon). Sew up the external oblique and skin.

FEMORAL HERNIA.

Etiology.—Before puberty both sexes equally, but rarely, affected. Commoner in women after puberty, because of the increased width of the pelvis opening out the crural canal.

Femoral Hernia, *continued.*

Anatomy.—Emerges through the crural canal. Crural canal is innermost part of the femoral sheath. Femoral sheath is formed by the junction of: (1) The fascia transversalis in front of the vessels; (2) The fascia iliaca behind the vessels.

RELATIONS OF THE CRURAL CANAL and of the neck of a femoral hernia.

In Front.—Transverse thickened part of the femoral sheath—deep crural arch. Poupart's ligament—superficial crural arch. Iliac portion of fascia lata and cribriform fascia.

On Inner Side.—Gimbernat's ligament.

On Outer Side.—Femoral vein.

Behind.—Os pubis. Pectineus muscle covered by pubic portion of the fascia lata.

Above it is closed by subserous tissue—the septum crurale.

Below it abuts against the cribriform fascia, closing in the saphenous opening of the fascia lata.

COVERINGS.

1. Skin and fascia, etc.
2. Cribriform fascia.
3. Femoral sheath (transversalis fascia).
4. Subserous tissue (septum crurale).
5. Peritoneum.

NECK.—Situated at the crural ring.

In Front.—Poupart's ligament and inguinal canal with its contents.

Inner Side.—Gimbernat's ligament.

Behind.—Pubic part of fascia lata covering os pubis.

Outer Side.—Femoral vein.

RELATIONS TO VESSELS.—Femoral vein on outer side. Long saphenous vein enters below it. Epigastric artery to its outer side.

Obturator artery is given off as branch of the deep epigastric in one in four cases. Runs on the outer side of the sac between it and the femoral vein generally. Runs on the inner side of the sac by Gimbernat's ligament in one in seventy-five cases. In these only is there much danger of its division in herniotomy.

DIRECTION OF GROWTH.—After emerging from the saphenous opening, the hernia is turned upwards over Poupart's ligament by the attachment of the deep layer of the superficial fascia to the fascia lata along a horizontal line level with pubic spine.

Contents of a femoral hernia.—Small intestine (ileum)—generally. Omentum—sometimes. Large intestine when left-sided—rare. Ovary or Fallopian tube—occasionally.

Signs.—Swelling below inner end of Poupart's ligament. Reducible with a gurgle. Expansile impulse on coughing. Neck lies to the outer side of and below pubic spine; lies behind Poupart's ligament and inguinal canal. When it rises above Poupart's ligament it obscures the fold of the groin, and lies superficial to the inguinal canal.

Diagnosis.—May be difficult in fat women, when hernia is irreducible.

INGUINAL HERNIA and INFLAMED LYMPH-GLAND (*vide supra*).

LIPOMA.—Long history and stationary size.

PSOAS ABSCESS.—Relation to iliac swelling. Descends behind the femoral sheath.

SAPHENOUS VARIX.—Veins below are generally varicose. Often fills after emptying in spite of pressure over crural canal.

Treatment.

TRUSS is less satisfactory than in inguinal hernia, because it presses on the vessels, and because the pad lies over the pubic bone, where pressure causes pain and rubbing.

OPERATION is less satisfactory because it is difficult to close the crural ring.

METHOD.—Vertical incision. Isolation of sac. Open sac and empty. Tie neck of the sac high up. Either:

1. Sew together iliac part of fascia lata to pubic part of fascia lata, and so close the saphenous opening; or
2. Detach pubic origin of the pectineus, and sew it to Poupart's ligament; or
3. Fix Poupart's ligament to the pubic bone by metal staples.

UMBILICAL HERNIA.

Varieties.

CONGENITAL.—Hernia protrudes into base of umbilical cord. Gut may be tied and cut with the cord. Very rare.

INFANTILE.—Hernia through a stretched umbilical cicatrix. Tends to spontaneous recovery. Treated by a binder.

ACQUIRED.—Hernia through linea alba above or below the navel. Generally in stout multiparous women. Sac is very thin and lobulated. Contents, which generally include part of the transverse colon and omentum, are very adherent. Much fat is developed: (1) Outside the sac wall; (2) In the omental contents.

Treatment.—Support by a binder or truss, or operate.

METHOD OF OPERATION.—Vertical incision. Open sac. Remove omentum and separate adhesions. Isolate and cut away the sac. Cut the margins of the neck of the sac so as to open the rectal sheaths. Sew the posterior sheaths of recti together. Sew the recti together. Sew the anterior rectal sheaths together.

OTHER VARIETIES OF HERNIA.

Ventral Hernia.

IN LINEA ALBA ABOVE NAVEL.—Generally a protrusion of subserous fat only. Small in size, but very painful.

TREAT by removal of fat and sewing up hole in linea alba.

Ventral Hernia, *continued.*

IN LINEA ALBA BELOW NAVEL.—After parturition. Tends to spontaneous recovery if supported by a belt.

In bad cases only, OPERATE. Sew edge of one rectus to the deep surface of the other. Sew edge of the other rectus to the superficial surface of the first.

Post-Operative.—Especially after suppurating appendicitis.

OPERATE on all young patients. Sew abdominal layers together one by one.

INSERTION OF A WIRE FILIGREE between the parietal layers makes relapse much less likely.

PATHOLOGICAL CONDITIONS OF HERNIA.

Varieties.

1. IRREDUCIBILITY.
2. INFLAMMATION.—Involves 1.
3. OBSTRUCTION.—Involves 1.
4. STRANGULATION.—Involves 1, 2, and 3.

1. Irreducibility.

DEFINITION.—Contents of a hernia cannot be returned into the abdomen.

CAUSES.

ADHESIONS OF THE CONTENTS TO THE SAC.

ADHESIONS OF THE CONTENTS TO EACH OTHER, so that a bulky mass larger than the neck of the sac is formed.

ADHESIONS OF THE WALLS OF THE SAC TO EACH OTHER, forming bands or constrictions. Follows attacks of inflammation. Occurs during obstruction and strangulation.

PRESENCE OF OMENTUM in the sac. From adhesions, fat accumulation, or cyst formation.

OCCURRENCE.—In order of frequency in umbilical, femoral, and large scrotal herniæ.

SIGNS.—A swelling at one of the hernial orifices. Gurgling may be felt and heard. Portion of the contents can often be reduced.

DIAGNOSIS.—From the following conditions (*vide supra*): Inflamed glands—Lipoma of the parietes or cord—Abscess (e.g., psoas abscess).

DANGERS.—Obstruction—Strangulation.

TREATMENT.

FIRM PRESSURE.—In bed by bandages and ice-bags. By a special truss—the hinged-cup truss.

OPERATION for radical cure.

Contra-indication for operation: In old patients with no symptoms. Very large hernia.

SUPPORT by a bag truss.

2. Inflammation of a Hernia.

DEFINITION.—Inflammation of the peritoneal sac and of the contents of a hernia.

CAUSES.—Injury, e.g., by taxis. Escape of bacteria through the gut wall. Strangulation.

ANATOMY.—An exudation from the sac wall and from the peritoneum covering the contents. Nature of the exudation is serous, plastic, or purulent.

SIGNS.—Those of external inflammation. Heat, redness, tenderness, and œdema over the hernia. Hernia is irreducible. Temperature rises. Some constipation and vomiting.

RESULTS.—Abscess forms and bursts externally. Strangulation may supervene. Irreducibility generally remains.

TREATMENT.—Rest in bed with fomentations. Incision and drainage if suppuration occurs. Radical operation cannot be done until the septic inflammation is over.

3. Obstruction of a Hernia.

DEFINITION.—Lumen of the gut in a hernia is obstructed without strangulation of its coats.

CAUSES.—Presence of solid matter in the gut. Specially liable to occur in umbilical hernia, which often contains the transverse colon. Complicated matting together of the gut interferes with its peristaltic action.

SYMPTOMS.—The hernia is irreducible and larger than usual. Constipation, vomiting, and colicky pains—occurring together in attacks lasting several hours or days.

RESULTS.—Recovery from early attacks as a rule. Strangulation ends an attack ultimately.

TREATMENT.—Rest in bed with low diet. Pressure and an ice-bag over the hernia. Copious enemata. Radical cure when the obstruction is over.

4. Strangulated Hernia.

DEFINITION.—Constriction of the contents of a hernia to such a degree as to stop the circulation in them.

CAUSES.

- (*a*) EXISTENCE OF A TIGHT STRANGULATING BAND or constriction. The neck of the sac itself in congenital inguinal cases. The matted tissues at the internal abdominal ring in acquired inguinal cases. Gimbernat's ligament in femoral cases. Bands of adhesion inside the sac.
- (*b*) THE FORCING DOWN OF AN EXTRA PORTION of the viscera through the neck of the sac.
- (*c*) ADHESIONS AND KINKING of coils of gut resulting from inflammation.

Strangulated Hernia, *continued.*

PATHOLOGY.

(*a*) THE GUT.

First Stage.—Stasis of the circulation, with venous engorgement. Colour is dark red or purple. Great thickening of the gut wall from œdema. Distention of the lumen by gas. Escape of bacteria, viz., the staphylococci and bacillus coli, through the gut wall. Small ecchymoses may occur. Peritoneum is smooth and shiny. Vessels can be emptied by pressure and refill easily.

Second Stage.—Thrombosis. Peritoneum is covered by plastic lymph and has lost its lustre. Colour is a dark purple. Vessels cannot be emptied by pressure. Free passage of bacteria through the gut wall.

Third Stage.—Gangrene. Gut is a slate-grey or black. May be perforated, or gives way on handling.

At the Site of Strangulation.—A pressure ulcer occurs from the outside. Bacteria escape through this. Perforation may occur before, during, or after reduction.

Above the Strangulation.—Distention of the gut from obstruction. Paralysis and peritonitis occur later if the condition has long continued.

(*b*) OMENTUM.—Undergoes similar changes, but gangrene is much rarer.

(*c*) SAC.—Undergoes the changes of peritonitis. Inflammation with copious serous exudation. Exudation becomes blood-stained and offensive. Plastic lymph may be deposited on its surface. Sloughing from the virulence of bacterial action. Inflammation heralded by œdema of tissue outside the sac. Sac bursts and a fæcal fistula results.

Anatomical Varieties of Strangulated Hernia.

SIMPLE ENTEROCELE.—The common form.

EPIPLOCELE.—Rarely strangulated unless bowel occurs in the hernia also.

RICHTER'S HERNIA.—A part only of the circumference of a piece of gut is strangulated.

LITTRÉ'S HERNIA.—A blind diverticulum only is strangulated, e.g., the appendix or Meckel's diverticulum.

Signs.

AT FIRST.—The hernia becomes hard or tense feeling. Irreducible. No impulse on coughing. Tender or painful. History of recent enlargement.

LATER.—Skin becomes inflamed and œdematous. Dark colour with emphysema if patient survives.

THE ABDOMEN.—Severe spasmodic pain referred to the navel. Tenderness and rigidity markedly absent at first. Distention and tympanites (from meteorism). Signs of peritonitis occur late in the case.

Symptoms.

SHOCK.

MOST MARKED IN: Pure enterocele—Partial enterocele (Richter's hernia)—When a hernia becomes strangulated on its first occurrence (children, congenital cases).

LEAST MARKED IN: Old-standing cases—Large herniæ—Cases preceded by irreducibility and inflammation—Epiplocele.

Pulse slow and weak, then rapid and irregular. Temperature subnormal, but rising with the onset of inflammation. Feeling of faintness.

PAIN.—Referred to the region of the umbilicus. Also over the region of the hernia. Occurs as spasms of colic supervening on a constant abdominal pain. Becomes less on the onset of gangrene.

VOMITING.—First of food. Later of bile and small intestine contents. Lastly of foul stercoraceous fluid.

CONSTIPATION.—Absolute from the first in most cases. Caused by: (*a*) Obstruction of the lumen of the gut; (*b*) Paralysis of the gut by nerve shock; (*c*) Peritonitis (at last).

Not present at first: In strangulation of the gut high up, when the lower bowel may empty itself.

Not present at all: In epiplocele, or any case when a viscus (e.g., ovary) other than gut forms the contents of the hernia. In partial enterocele (rare).

TOXÆMIA.—Occurs after the case has persisted some time. Shrunken face, hollow eyes, anxious expression. Temperature becomes again subnormal. Drowsiness, delirium, and coma.

LATENCY OF THE SYMPTOMS is often well marked and very important. Occurs especially in elderly patients, and in old-standing hernias. Shock may be absent entirely. Only symptoms present may be constipation, a little vomiting, and local tenderness over the hernia.

Gangrene of the gut in such cases may be signified by: Relief of pain in the abdomen. Relief of tension and tenderness in the hernia. Occurrence of œdema over the hernia.

N.B.—Gangrene of the gut may occur within a few hours of strangulation.

Treatment.

TAXIS.—Should only be used in the following conditions:—

1. When the hernia has only just appeared, e.g., in congenital hernia when the contents come down suddenly and are at once strangulated.
2. When the hernia has been reducible a short time previously.
3. Within a few hours of the onset of strangulation.
4. For a short time (ten minutes).
5. With the utmost gentleness.
6. Under an anæsthetic, which may be prolonged for the operation of herniotomy.

Strangulated Hernia—Treatment, *continued.*

OPERATION without any unnecessary delay. Directly the diagnosis is clear or even probable. In all except the few cases when taxis is permissible and successful.

DANGERS OF DELAY in operation.

Strangulation has no tendency to natural recovery, except the very rare occurrence of a natural fæcal fistula.

SEPSIS begins to occur with strangulation, by an escape of bacteria through the congested gut.

GANGRENE OF THE GUT may result in a few hours.

PARALYSIS OF THE GUT, with incapacity for recovery, may result from prolonged distention.

THE GENERAL CONDITION of the patient becomes worse and not better from delay, because of: (1) Want of food; (2) Want of sleep; (3) Loss of fluid by vomiting; (4) Rapidly increasing septic absorption.

Operation for Relief of Strangulated Hernia.

PRELIMINARIES.—Wash out the stomach if vomiting is severe. Or anæsthetize by local or spinal anæsthesia.

EXPOSURE OF HERNIAL CONTENTS.—Cut down to the sac. Open sac carefully and let out contained fluid.

DIVISION OF THE STRANGULATING AGENT.—Usually found at the neck of the sac. Occasionally in the body of the sac or in the contents. In oblique inguinal herniæ, cut outwards at the neck of the sac. In femoral hernia, cut inwards Gimbernat's ligament. In all others, cut any tense band that may be felt. Make the incision as small as possible: just sufficient to free the contents.

EXAMINATION OF CONTENTS.—First pull the contents down further, so that the actual site of strangulation can be examined. Decide whether the contents, especially the gut, are viable.

SIGNS OF VIABILITY of the gut or other viscus.—The peritoneum is smooth and shiny. Blood-vessels can be emptied by pressure and refill.

SIGNS OF THE GUT NOT BEING VIABLE.—The peritoneum has lost its lustre. May be covered by lymph. Colour is deep purple or slate. Vessels cannot be emptied by pressure.

TREATMENT OF THE GUT and other hernial contents.

ANY LIMITED ULCER at the site of strangulation, or

ANY LIMITED PATCH OF GANGRENE

Should be infolded by sewing the neighbouring healthy gut over them.

VIABLE GUT should be returned into the abdomen.

REDUNDANT OMENTUM should be ligatured and removed.

NON-VIABLE GUT should be excised.

Cutting well above and below the site of strangulation.

Immediate anastomosis of the healthy ends, except in the following:—

Sew the ends together in part of their circumference, and tie in a tube at the remaining part, pushing the junction just within the abdomen: (1) When great distention exists; (2) When the patient's condition is desperate.

RADICAL CURE should complete the operation, except when the patient's condition is grave, or when gangrene and sepsis are evident. In this case drain through the opening in the parietes.

AFTER-TREATMENT.—Turpentine and soap enemata until natural evacuations occur. Continuous rectal infusion of saline fluid in cases with marked collapse or peritonitis. Avoid feeding by mouth until the bowels have acted.

Complications after Operation.—Intestinal obstruction from paralysis of the gut. Peritonitis. Acute enteritis, passage of blood and mucus.

Taxis.—Except in the cases mentioned above, this is much more dangerous than operation, and is often the cause of a fatal result following an operation.

DANGERS.

1. IT MAY DAMAGE THE SAC AND ITS CONTENTS, especially the gut. Causes œdema and swelling. Produces hæmorrhage into the sac and into the gut wall. May rupture the gut.
2. MAY FORCE INFLAMED OR GANGRENOUS GUT BACK into the abdomen. Hastens the transudation of bacteria through the gut wall. Hastens gangrene. Causes unnecessary suffering and delay. Makes the subsequent operation more difficult and dangerous.
3. IT MAY REDUCE THE HERNIA WITHOUT RELIEVING THE SYMPTOMS.
    - (*a*) By the reduction of a ruptured or gangrenous gut.
    - (*b*) By causing peritonitis from the reduction of septic contents into the abdomen.
    - (*c*) By forcing the sac with its strangulated contents back into the abdomen—"*reduction en masse.*"
    - (*d*) By forcing the hernial contents into a pouch leading out of the main sac, such as occurs in an interstitial hernia—"*reduction en bissac.*"
    - (*e*) By forcing the contents through a rupture in the neck of the sac.
    - (*f*) By reducing contents whose strangulation is due, not to the neck of the sac, but to a band or kinking of the contents themselves.

CHAPTER XXXIX.

DISEASES OF THE RECTUM AND ANUS.

CONGENITAL MALFORMATIONS OF THE RECTUM.

1. ABSENCE OF RECTUM AND ANUS.—Colon ends at the pelvic brim. The pelvis is narrow and ill-developed. No bulging occurs in perineum when child cries.
2. RECTUM IS PRESENT, BUT THE ANUS IS ABSENT.—Bulging occurs in the perineum on crying.
3. ANUS AND ANAL CANAL ARE PRESENT, BUT DO NOT JOIN THE RECTUM.—Finger feels a partition about one inch above anus.
4. A STRICTURE MAY EXIST where anal canal joins rectum.

   TREATMENT.
   - (*a*) Dilate a stricture by daily passage of bougies.
   - (*b*) Perforate a septal division, and then dilate.
   - (*c*) Where anus is imperforate: Cut upwards in mid-line for two inches; bring rectum down, open, and sew to skin.
   - (*d*) If the rectum cannot be reached from the perineum: perform inguinal colotomy.
5. RECTUM OPENS INTO OR COMMUNICATES with bladder, vagina, urethra, or vulva.

   TREATMENT by a plastic operation in slight cases, after preliminary colotomy to divert the fæces.

PROCTITIS, OR INFLAMMATION OF THE RECTUM.

Causes.—Foreign bodies—Fish-bones or other hard material in fæces—Polypus or tumour of the rectum—Piles, fissure, or fistula—Prolapse—Dysentery—Syphilis—Tuberculous ulceration—Gonorrhœa—Parasites: Threadworms (in children), bilharzia (in patients from Africa.)

Symptoms.—Bearing-down pain. Painful tenesmus. Feeling of weight and fullness in perineum. Discharge of mucopus from the anus.

Treatment.

1. Ascertain and remove the cause, e.g., piles, fissure, or foreign body.

2. Copious hot-water enemata. Lead and opium lotion or suppositories in acute stage. Lot. protargol, gr. j ad ℥j, in later stages.
3. For threadworms: Purge, and inject with infus. quassiæ.
4. For bilharzia: Scrape away the adenomatous polypi which contain the eggs.
5. Incision of sphincter if other means fail.

ISCHIO-RECTAL ABSCESS.

Anatomy.—Ischio-rectal fossa is triangular in vertical section. Outer wall is formed by ischium clothed by obturator internus. Inner wall is formed by the rectum clothed by levator ani. Lower wall is formed by the skin. In front is the triangular ligament. Behind is the border of the gluteus maximus.

Sources of Infection may be: Through any of the three walls or from in front.

1. Through rectum by ulceration or perforation of foreign body.
2. Through skin by wound abrasion or sebaceous gland.
3. Through the outer wall from disease of pelvis.
4. From the urethra in front.

Varieties.

1. CELLULITIS.—Possibly gangrenous. Diffuse cellular infection from the bowel or skin. Generally in feeble old people.
2. ACUTE ISCHIO-RECTAL ABSCESS. Bacillus coli infection from the bowel, or a pyogenic infection from the perineum.
3. CHRONIC ABSCESS.—Generally tuberculous in origin.

Symptoms.—Those of local inflammation. Brawny swelling in the perineum. Hot, very painful swelling, felt per rectum. Defæcation is very painful. Bursts either through the skin or into the bowel, or in both directions at once. Often results in a fistula.

Treatment.

ACUTE CASES.—Open freely from the perineum and drain. If an opening exists into the bowel, cut right through the rectal wall and sphincter.

CHRONIC CASES.—Try scraping and stuffing with iodoform. Liability to sepsis makes healing bad. Colotomy has often to be performed.

FISTULA IN ANO.

Definition.—Any sinus near the anus.

Varieties.

1. COMPLETE.—Sinus opens into the rectum internally; on to the perineum externally.
2. INCOMPLETE EXTERNAL.—A perineal sinus not opening into the rectum.
3. INCOMPLETE INTERNAL. A sinus opening into the rectum, but not externally.

Fistula in Anô, *continued.*

Causes.—The causes of proctitis. Ischio-rectal abscess. Ulceration or stricture low down in the rectum: malignant, tuberculous, or syphilitic.

Anatomy.—A suppurating or granulating sinus communicates with one or both openings. It runs outside the bowel entirely, or more frequently perforates the gut obliquely, so running some distance between mucous membrane and muscular coat. May almost encircle the bowel. The internal opening is usually above the internal sphincter.

IN CHRONIC CASES: Dense cicatricial tissue forms round the sinus. Multiple diverticula branch off in various directions, especially upwards between the sacrum and anus. Both internal and external openings become multiple.

Symptoms.—Pain, especially at defæcation. Purulent discharge from a sinus, or from anus with or after the motion.

Treatment.

PRELIMINARY.—Saline aperients for several days. Opium pill, gr. j, twelve hours before. Enema just before operation.

OPERATION.—Pass a director up the fistula. Open it completely from end to end. Scrape out all granulations and cut out scar tissue. Cut through all tissue between rectum and the fistula, including the sphincter ani. Open up all secondary channels and sinuses. Pack deeply with oiled gauze. In cases in which the fistula is superficial to the sphincter, the latter should also be deeply incised as in the case of a fissure.

IN CHRONIC OR COMPLICATED CASES.—Either excise the lower end of the rectum, retaining the sphincter (Kraske's operation); or perform an inguinal colotomy.

FISSURE IN ANO.

Definition.—A longitudinal ulcer in the anal margin.

Situation, etc.—Usually single and in mid-posterior line where the anus is most firmly fixed to the coccyx.

Below the fissure is a "SENTINEL PILE," which is a tag of mucous membrane that has been torn down by a scybalous mass, so forming the ulcer.

Rather commoner in men than women.

Symptoms.—Severe burning pain during and after defæcation, with great tenesmus. Fæces may be streaked with blood.

Treatment.—Laxatives and copious hot enemata. Cocaine suppositories and the ung. gallæ c. opio.

OPERATIVE.—Under an anæsthetic: (1) Dilate the sphincter; (2) Remove all piles, especially "the sentinel;" (3) Cut out the ulcer, making the incisions extend right through the sphincter so as to paralyze it temporarily.

FIBROUS STRICTURE OF THE RECTUM.

Etiology.—Elderly patients. Usually women. Follows (1) Ulceration due to dysentery, tubercle, or syphilis; (2) Cellulitis contracting outside the gut; (3) Other conditions of proctitis, e.g., that due to gonorrhœa or piles.

Anatomy.—Stricture is situated two or three inches from the anus. It may be (1) Annular and smooth; (2) Long and irregular; (3) Tied to the sacrum. The gut above is hypertrophied, dilated, and ulcerated. The gut below is often ballooned.

Symptoms.—Alternating attacks of constipation and diarrhœa. Attenuated moulded fæces, with scanty blood and mucus. Chronic intestinal obstruction. Ischio-rectal or pelvic abscesses may form by rupture or infection through a stercoral ulcer. Large fæcal abscesses may form in the buttock, and cause death or chronic fistulæ.

Treatment.

DILATATION by graduated bougies, when these can be borne.

EXCISION of the stricture when it is short and the bowel above and below healthy and free.

SACRAL OR INGUINAL ANUS in the worst cases.

SYPHILIS OF THE RECTUM AND ANUS.

Primary Chancre is rarely situated at the anus.

Secondary Condylomata are very common round the anus.

Tertiary Gummata form a diffuse infiltration of the rectal wall. They are commoner in women than men. The disease begins in the submucous tissue. Ulceration soon follows, and is deep but comparatively painless. It may extend to the anus, sigmoid, vagina, perineum, or bladder, and form fistulæ. It usually results in an extensive stricture.

SYMPTOMS.—Some pain and rectal tenesmus. Discharge of blood and pus per anum. Later, the signs of chronic obstruction.

TREATMENT.

1. MERCURY AND IODIDES until all active disease has been arrested.
2. Temporary or permanent COLOTOMY.
3. In a very few cases when the stricture is short, EXCISION.

NEW GROWTHS OF THE RECTUM AND ANUS.

Polypus, or stalked adenoma, consists of simple gland tissue with more or less connective tissue connected to the submucous coat by a vascular pedicle. It is commonest in children. It may cause some rectal pain, irritability, or prolapse.

TREATMENT by ligature and removal.

New Growths of the Rectum and Anus, *continued.*

Papilloma.—May be localized or a diffuse growth, with a tendency to free hæmorrhage. It is of rare occurrence, and must be treated by REMOVAL.

Bilharzia Papilloma is a polypoid outgrowth of mucous membrane, bleeding freely, in which many bilharzia ova are found.

Epithelioma attacks the anus. Often in the site of an old fissure. It may form a warty mass, but soon becomes ulcerated. It is indurated, its edges hard and everted. It bleeds very freely, and has a copious foul discharge. Secondary deposits occur very early in the inguinal glands.

TREATMENT by early free removal, together with the glands in both groins.

Sarcoma is a rare disease, occurring in young patients (under thirty). It forms a rather diffuse growth in the submucous tissue which causes obstruction. Ulceration is absent or late.

It should be removed, but recurrence is likely.

CARCINOMA OF THE RECTUM.

Etiology.—A little commoner in men than women, and occurs generally after middle life.

Anatomy.—It begins in the mucous membrane, and thence spreads (1) Into the lumen of the gut; (2) Up and down the gut; (3) Out through the gut wall in neighbouring structures.

ITS MACROSCOPIC STRUCTURE may be of three types:—

1. A FUNGATING FRIABLE MASS projecting into the bowel.
2. AN ULCER with hard raised margin, of considerable extent.
3. A FIBROUS ANNULAR CONSTRICTION with comparatively little new growth or ulceration.

The bowel above and below shows the same changes as are described in the section on intestinal obstruction.

MICROSCOPICALLY it is always a columnar-celled carcinoma, which often shows colloid degeneration, and in which the proportion of glandular and fibrous tissue varies. Thus, in the soft, rapidly growing kinds, the glandular tissue predominates, whilst the hard annular varieties consist chiefly of fibrous tissue.

Extension occurs outwards through the bowel wall, and may involve: (1) The cellular tissue; (2) The sacrum; (3) The pelvic vessels and nerves; (4) The vagina or uterus; (5) The prostate, bladder and ureters. In the case of the vagina or bladder a bimucous fistula may result by ulceration.

Metastasis is comparatively late and rare. It occurs: (1) In the lumbar glands; (2) In the liver and peritoneum; (3) In the inguinal glands when the anus has been involved.

Symptoms and Signs.

1. EARLY, BEFORE OBSTRUCTION.

   PAIN of a dull, dragging kind, with some tenesmus. This may be quite absent.

   BLEEDING, both with and between the motions.

   Passage of offensive blood-stained mucus.

   Rarely the motions may become narrow and moulded.

   DIARRHŒA alternates with constipation.

   ON EXAMINATION a mass is found which is either (*a*) Friable and fungating, or (*b*) Hard, with ulcerated surface and everted margins. The mass is not fixed in any way, and on straining the bowel descends as a whole.

2. ADVANCED, WITH OBSTRUCTION.—The signs and symptoms of chronic intestinal obstruction are added to the above. Great abdominal distention, with visible peristalsis. Pronounced constipation, with colicky pain, which is increased by purgatives. Pain and tenesmus are much more constant and severe.

   ON EXAMINATION, the lumen of the bowel is partially obliterated by a stricture or fungating mass.

3. INVOLVEMENT OF NEIGHBOURING STRUCTURES.—Severe constant or neuralgic pain is caused by extension to the sacrum and sacral nerves. Œdema, by involvement of the vessels. Gas or fæces may be voided from the urethra, and cystitis supervene from bladder extension. Hydronephrosis, pyonephrosis and uræmia, from blocking of the ureters. Gas and fæces may be passed from the vagina.

   An abscess may form in the pelvis, ischio-rectal fossa, or buttocks, and give rise to one or many fistulæ, which communicate with the gut above the growth.

   ON EXAMINATION, the growth is fixed and the bowel does not come down at all on straining. This fixidity may be only partial, e.g., to the bone behind or vagina in front, or it may be complete, the whole bowel circumference being involved.

4. LATE CACHEXIA with secondary growths.—This stage is often never reached, the patient dying either from intestinal obstruction or sepsis. Masses of glands may be felt in lumbar regions. The liver becomes large and nodular, and ascites develops.

Course.—Varies very much. In young patients and with soft fungating growths the whole course may be only one year. In older patients and with fibrous growths it may last five years.

Diagnosis.—This is usually easy if the case is examined locally. In the early stages IT IS IMPOSSIBLE to diagnose cancer from piles, prolapse, polypus, or fistula WITHOUT A RECTAL EXAMINATION.

IN PILES there is no new growth, but merely swollen veins, but it is important to remember that piles often complicate cases of cancer, so that the inside of the bowel should be examined in every case of piles.

Carcinoma of the Rectum—Diagnosis, *continued.*

IN FISSURE the pain is much more severe, and is almost unbearable on digital examination. A linear ulcer with sentinel pile is evident, and the inside of the bowel is healthy.

IN POLYPUS the mass is small and pedunculated, and the mucous membrane healthy.

IN FIBROUS STRICTURE the parts are indolent and there is no new growth. Bleeding on examination is absent or very slight.

IN SYPHILITIC DISEASE the gummata are soft, painless, and non-vascular. The ulcers ragged, undermined, and not at all indurated.

IN PROLAPSE AND INTUSSUSCEPTION inspection and palpation will make the case clear.

THE SIGMOIDOSCOPE is useful for cases of cancer occurring out of reach of the finger (three to six inches from the anus).

Treatment.

CASES UNSUITED FOR EXCISION are nearly 70 per cent of all seen. Growth is fixed to the bladder, prostate, or ureters, or to the sacrum high up. Extension into the cellular tissue or glands. Metastatic growths. Condition of age and debility.

TREATMENT.—Soft food with little débris, avoiding fruit, vegetables, etc. Bowel washed out through a soft tube daily. Olive oil enemata.

COLOSTOMY for obstruction.

LOW OPERATION.—Perineal excision.

INDICATIONS.—For cases in which the disease extends within one inch of the anus, but not higher than the finger can reach.

METHOD.—The anus is excised with the lower end of the bowel well above the growth. The wound is either plugged round a central tube without any stitching of the gut to the skin, or a few anchoring stitches may be used.

HIGH OR TRANS-SACRAL EXCISION.

INDICATIONS.—For cases in which the sphincter ani can be preserved. The disease ought not to extend higher than five inches from the anus, i.e., above the third piece of the sacrum.

METHOD.—A preliminary colostomy ought always to be done: (1) To ascertain presence of other growths and the extent of the rectal disease; (2) To divert the fæces from the wound; (3) To relieve obstruction.

After excising the coccyx the incision is produced up over the sacrum, and the lower part of this removed obliquely or transversely, keeping below the third piece; otherwise the sacral plexus will be injured.

The bowel is isolated, usually the peritoneal cavity is opened and packed. As much of the posterior cellular tissue and glands as possible are removed from the pelvis.

The growth is excised, and the ends of the bowel above and below are sutured. This can be done in some cases by invaginating the proximal through the distal end and through the anus, and then by a circular continued stitch.

COMBINED METHOD.

INDICATIONS.—May be used for all cases in which the disease extends above the third sacral vertebra.

METHOD.—The bowel is freed, and the vessels in the sigmoid mesocolon tied from the abdomen, and then the loop is pulled down, and the anastomosis made by the sacral route.

HÆMORRHOIDS OR PILES.

Definition.—Varicose veins of the anal region.

Causes.—Pressure of the blood in the portal system. Absence of valves in the portal veins. Anastomosis between the portal and systemic system that occurs between the superior, middle, and inferior hæmorrhoidal veins. Any portal congestion, cirrhosis of the liver, or intra-abdominal tumour. Alcoholism and chronic constipation.

Specially common in young men and middle-aged pregnant women.

Varieties.—External and internal.

External Piles are covered by skin.

CONSIST of a central vein covered by a tag of hypertrophied skin.

SYMPTOMS are slight. Pruritus and some pain. Inflammation and thrombosis or suppuration are frequent.

TREATMENT by hamamelis ointment and careful washing. Fomentations when inflamed. Excision must be sparingly done, so as to avoid excessive cicatrization.

Internal Piles occur inside the sphincter and may extend two inches up the bowel.

CONSIST OF varicose veins covered by hypertrophied mucous membrane. They form a series of fleshy, purple masses inside the anus, some being thick, firm, and fleshy, and others thin-walled and easily bleeding.

SYMPTOMS.—Pain and dull aching after defæcation. Very sharp pain is due to a fissure.

BLEEDING after defæcation, the blood being spattered over the pan.

Mucoid discharge, with tendency to prolapse.

COMPLICATIONS.

INFLAMMATION and thrombosis. If suppurative, may cause portal pyæmia.

STRANGULATION and sloughing by pressure of the sphincter on prolapsed piles.

PROLAPSUS RECTI.

FISSURE IN ANO.

Internal Piles, *continued.*

TREATMENT.

GENERAL.—Saline aperients, and plenty of exercise.

LOCAL.—Washing after defæcation. Astringent ointments, e.g., the ung. hamamelis or ung. gallæ c. opio.

OPERATIVE.—Where bleeding or pain are severe, and where no serious permanent portal congestion, e.g., cirrhosis, exists.

PRELIMINARY.—Purgative, e.g., castor oil following salines. Discontinue these forty-eight hours before. Enema twelve hours before operation.

Lithotomy position. Four methods: (1) Clamp and cautery; (2) Ligature and excision; (3) Crushing; (4) Whitehead's operation, or an excision of the whole pile-bearing area of mucous membrane. This is liable to set up pelvic inflammation.

Bowels are kept confined for six days after.

PROLAPSE OF THE RECTUM.

INCOMPLETE.—Is commonly associated with piles in adults, when only the mucous membrane protrudes.

COMPLETE.—Is common in children, when all the coats extrude.

Causes.—(1) Relaxed conditions of the tissues; (2) Constant straining, e.g., large prostate; (3) Diarrhœa or worms; (4) Chronic constipation, piles.

Signs, etc.—Protruding mass at the anus, with central orifice communicating with the lumen of the rectum, its walls joining the skin at the anal margin. Mucoid discharge and bleeding. Incontinence of fæces when it is chronic. Strangulation and sloughing are very rare.

DIAGNOSIS from piles, polypus, and intussusception.

Treatment.

PALLIATIVE.—Removal of any cause of straining or any rectal irritation, e.g., worms or piles.

ASTRINGENT LOTIONS, defæcation in a lateral position the last thing at night; a pad soaked in the lotion is tied on with a T bandage over the anus.

OPERATIVE.

Removal of the mucous membrane in incomplete prolapse.

Linear scarification, injection of astringents or of paraffin.

Excision of the whole projecting tumour, with suture of the bowel in complete cases.

Colopexy, or suture of the sigmoid flexure to the parietes, in a very few relapsing cases.

CHAPTER XL.

AFFECTIONS OF THE LIVER AND BILE-DUCTS.*

WOUNDS OF THE LIVER.

Non-penetrating.

CAUSES.—Buffer accidents—Running over—Blows, kicks, falls, etc.—Fracture of ribs.

PREDISPOSING.—Fatty or congested liver.

VARIETIES.

CONTUSION.—Ecchymosis. Glisson's capsule is unruptured.

RUPTURE.—Glisson's capsule ruptures. A portion of liver may be detached completely.

SITUATION, ETC.—Right lobe most common. Antero-posterior laceration. May be transverse in superior surface if caused by doubling up of liver.

SYMPTOMS.

SHOCK, from violence of accident.

COLLAPSE, from internal hæmorrhage.

BRUISE, from point of application of force over the liver.

PAIN over liver down to navel and up to scapula or ensiform cartilage.

PERITONEAL HÆMORRHAGE.—Abdomen is held rigid. Shifting dullness appears in the flanks ; most marked on the right side. Pulse becomes increasingly rapid. The distention and dullness increase rapidly.

URINE may contain bile or sugar.

PULMONARY EMBOLISM occasionally results from liver tissue forming an embolus.

FRICTION SOUNDS over the liver may be heard later.

TREATMENT.—Usual treatment for shock. Open abdomen if hæmorrhage is evident, or collapse continues. Incise through linea alba. Stitch up liver, or pack with gauze. Purse-string sutures may be used for small wounds. Incision may be enlarged by cutting round costal margin.

Penetrating or External Wounds.—Stabs—gunshot—lacerated, etc. (impaled). Much rarer than rupture.

SYMPTOMS.—Shock and collapse.—External or internal hæmorrhage (rapidly fatal)—Escape of bile—Vomiting—Hiccough.

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* Waring, "Diseases of the Liver, Gall-bladder, and Biliary System."

Penetrating or External Wounds of the Liver, *continued.*

LATER SYMPTOMS.—Tympanitic distention of abdomen—Rigors—Delirium—Peritonitis—Secondary hæmorrhage—Abscess.

DIAGNOSIS from position, direction, and depth of wound.

TREATMENT.—If hæmorrhage and shock increase, open abdomen and plug or suture. In gunshot wounds, remove missile if possible.

WOUNDS OF THE GALL-BLADDER AND BILE-DUCTS.

CAUSES and symptoms as in wounds of liver, but escape of bile is more marked. Bile soon makes its appearance in the urine.

RAPID DEVELOPMENT OF PERITONITIS.

TREATMENT.

Complete exposure by laparotomy.

WOUND OF GALL-BLADDER.—Sew up if small, and close abdomen. Sew to abdominal incision if large, or remove gall-bladder.

WOUND OF CYSTIC DUCT.—Cholecystectomy.

WOUND OF HEPATIC DUCTS.—Tie ends of duct.

WOUND OF COMMON BILE-DUCT.—Tie open ends. Cholocystenterostomy.

RUPTURE OF GALL-BLADDER OR DUCTS.

CAUSES.—Traumatism: Kick, blow, run-over, etc. Especially when gall-bladder is distended and a calculus is present—Ulceration from calculus—Typhoid fever—Tubercle—Ascaris.—Malignant disease (very rare)—Dilatation from blocking duct.

PATHOLOGY.

IF SUDDEN AND EXTENSIVE: General peritonitis results rapidly.

IF GRADUAL: An encysted fluid collection amounting to several gallons may result.

Continuity of bile-duct may be restored in few weeks in latter case, and this has been proved experimentally.

SYMPTOMS.—Either immediate septic peritonitis, or localized peritonitis, followed by fluctuating tumour—Jaundice—Bile in urine—Clay fæces.

DIAGNOSIS is that of acute peritonitis associated with traumatism, jaundice, or history of gall-stone colic.

TREATMENT.

In SUBACUTE OR OBSCURE CASES.—Keep in bed. If localized swelling occur—laparotomy.

IN ACUTE CASES.—Immediate laparotomy. Details as in last case (penetrating wounds). A septic gall-bladder should always be drained, or removed.

GALL-STONES.

Causes.

1. ANY OBSTRUCTION TO FREE OUTFLOW OF BILE, e.g., duodenal catarrh—Constipation—Tight-lacing, etc.

2. ANYTHING IMPEDING THE ABDOMINAL RESPIRATORY MOVEMENTS which hasten the flow of bile, e.g., Pregnancy—Abdominal tumour—Sedentary habits—Tight corsets.
3. PRESENCE OF MICRO-ORGANISMS: *Bacillus coli communis*, *Bacillus typhosus*. These set up catarrh, and excess of cholesterin crystals and bilirubin are formed, with clump of bacilli as nucleus.
4. FOREIGN BODY (rare): Needle — Ascaris — Distoma — Hydatid, etc.
5. EXCESS OF CARBOHYDRATE IN DIET: Diminution of bile acids, which keep cholesterin in solution. This would account for greater frequency in Germany, and in elderly females.

Seat of Formation.—Generally in gall-bladder. May be found in cystic or common ducts—more rarely in hepatic ducts.

Structure.—Thin layers: Nucleus—Middle and outer layer.

NUCLEUS consists of: Clump of bacteria—Mucus—Blood-clot—Casts of bile capillaries, or foreign body, or crystals of $CaCO_3$.

MIDDLE LAYER.—Laminated layers of cholesterin crystals.

EXTERNAL LAYER.—Bilirubin combined with a calcium salt.

Varieties.—Compounds of cholesterin and bilirubin-calcium. Either substance nearly pure. Latter in calculi formed in liver.

Occurrence.—In 4 per cent of all males; in 20 per cent of all females; in about same proportion, i.e., 1 in 5, they give rise to symptoms.

Age-occurrence.—Unknown in children. Common in mid-life—30–50. Commonest in old age—but often latent.

Great frequency and latency in old age is probably due to degeneration of muscular tissue of gall-bladder and ducts.

Symptoms.

INDEFINITE PAINS—Fullness in right hypochondrium and epigastrium.

PAROXYSMS OF COLIC.—Begin in hypochondriac and epigastric regions. Spread towards navel and right scapula. Generally begin in daytime, during exercise. Last a few minutes to several hours. Collapse, sickness, or vomiting.

SICKNESS AND MALAISE.—Quite independent of colic.

VOMITING.—At end of paroxysm. Bile-stained vomit. Calculus is rarely vomited.

COLLAPSE.—Feeble pulse. Clammy skin. Low temperature.

CALCULI IN FÆCES.—Absence in some cases accounted for by stone having failed to reach the intestine.

JAUNDICE.—Usually occurs 12–36 hours after colic. Skin, urine, conjunctiva, are yellow. Fæces are clay-coloured and offensive. Rarely, and in old people, jaundice occurs without preceding colic. Does not occur when stone is in cystic duct.

Is partial, i.e., fæces contain bile when obstruction is partial, or when fistulous communication exists.

Gall-Stones—Symptoms, *continued.*

TEMPERATURE.—Sometimes rigors, with temperature 103° or more, occur after colic. Probably due to septic infection at seat of ulceration.

Fever may be of an intermittent type, associated with catarrh of bile-ducts and an impacted calculus. May be remittent, and lead on to septicæmia or septic endocarditis. Associated with suppuration in bile-ducts.

Physical Signs.

ENLARGEMENT OF LIVER.—Slight—tender.

ENLARGEMENT OF GALL-BLADDER at ninth costal cartilage. Smooth and rounded. Points to right pubic spine. Moves with respiration. Can be moved laterally. May be due to mass of calculi, mucus, or pus.

This enlargement of the gall-bladder indicates blocking of the cystic duct, and there is in this case only slight jaundice.

WHEN THE COMMON DUCT IS BLOCKED, and in most chronic or recurrent cases, the gall-bladder is contracted.

If a stone produces marked jaundice it does not cause enlargement of the gall-bladder, and vice versa.

Diagnosis.—Usually follows from association of colic with jaundice, and there may be palpable gall-bladder or stone in fæces.

From RENAL COLIC.—In this: Pain radiates into pelvis and thigh; vesical irritation, crystals in urine.

LEAD COLIC.—In this: Chronic constipation, blue line, etc.

GASTRIC ULCER.—In this: Relation of pain to meals.

CANCER OF HEAD OF PANCREAS.—In this: Fixed tumour, loss of flesh, deep jaundice associated with enlargement of gall-bladder.

MEMBRANOUS COLITIS.—In this: Casts and shreds of colon are passed.

ABSCESS OF LIVER, ASCARIDES, HYDATIDS.

ZIEMSSEN'S TEST of nature of tumour in region of gall-bladder.

Inflate large intestine with air.

If tumour is pushed up and resonance is below it = Gall-bladder.

If tumour is pushed down or back, resonance above and in front of it = kidney or perinephric tumour.

Clinical Groups.—(1) Those in which the stone passes and gives rise to colic and transient jaundice; (2) Those in which the stone becomes impacted and colic is not present, but further sequelæ result.

Impaction: Its Complications and Sequelæ.

1. IMPACTION OF STONE WITHOUT OTHER COMPLICATIONS.

In neck of bladder, or cystic duct; gall-bladder forms large fluid tumour.

In common bile-duct: Permanent jaundice, unless fistulous communication with gut is formed.

2. Impaction followed by Septic Sequelæ.—Angiocholitis and cystitis, with rigors. Empyema of gall-bladder. Hepatic abscesses. Perforation of bile-ducts and abscesses in neighbourhood. Passage of stone in peritoneal cavity. Septic peritonitis.
3. Impaction followed by Adhesion to Stomach or Gut, with formation of fistula and passage of stone. Often no symptoms.
4. Impaction followed by Ulceration and Intestinal Obstruction.
5. Impaction followed by Adhesion to and Ulceration through Anterior Abdominal Wall.
6. Obliteration of Gall-Bladder.
7. Ulceration followed by Cicatrization and Stenosis of Ducts.
8. Carcinoma resulting from irritation of stone.
9. Chronic Pancreatitis from impaction in the ampulla of Vater.

Treatment.—MEDICINAL AND DIETETIC.

For Colic.—Morphia, gr. $\frac{1}{4}$; cum atrop. sulph., gr. $\frac{1}{12}$; hot bath; large quantity of water (warm), with sod. bicarb. and sod. salicyl., to promote flow of bile; inhalation of chloroform. Olive oil or glycerin of doubtful utility.

For Removal of Stones (?)—Course of Carlsbad waters.

For Prevention of Stones.—Exercise. Diet.

SURGICAL TREATMENT is called for in :—

Enlarged Gall-Bladder.
Persistent Jaundice.
Recurrent Attacks of Colic.
Intermittent Fever.
Signs of Local Inflammation.
Peritonitis.

Stones in Bladder.—Cholecystotomy.

Stone in Cystic Duct.—Push back and remove through the gall-bladder, or incise wall of duct, remove stone, and sew up.

Stone in Common Bile-duct.—Push forward or back, or excise and sew up. If the stone is in the third part of the duct, this must be done by opening the second part of the duodenum and removing the stone through the posterior wall of the gut.

BILIARY FISTULA.

External.

CAUSES.—Gall-stones—Penetrating wounds of gall-bladder or ducts—Hepatic abscess (especially of traumatic origin)—Surgical treatment of cysts or abscess of liver—Cholecystotomy—Actinomycosis—Malignant disease—Congenital.

POSITION.—Usually at umbilicus or right hypochondrium. May be in hypogastrium or inguinal region.

SYMPTOMS.—Discharge of bile, mucus, blood, or pus. Quantity will depend on patency of ducts. If entire bile is discharged externally patient emaciates and dies.

Biliary Fistula—External, *continued.*

TREATMENT.—Keep sinus clean. Drain. Scrape and pack. Remove gall-bladder. Remove obstruction from common duct. Cholecystenterostomy.

PROGNOSIS.—Due to malignant disease, abscess in depths of liver, or impermeable stricture—bad. Otherwise—good.

Internal.

CAUSED by adhesion to and ulceration into: Stomach—Colon—Duodenum—Pleura—Pelvis of kidney—Vagina—Portal vein—Encysted peritoneal cavities. Or by malignant infiltration of same organs.

SYMPTOMS.—Gall-stones may: (1) Be vomited; (2) Be voided per anum; (3) Cause intestinal obstruction; (4) Cause encysted peritoneal collection of fluid.

ABSCESS OF THE LIVER.

Causes.

1. TRAUMATISM—chiefly pyogenic cocci. Blows on hypochondrium. Penetrating wounds: From outside—From stomach (fish-bones, pins).
2. GALL-STONES—chiefly *B. coli communis.* Direct ulceration from gall-bladder or ducts. Indirect infection, especially when stone ulcerates in the common duct.
3. PARASITES.—Ascarides—Creep up duct from duodenum—Especially in children. Die and act as septic foreign body. Hydatid cyst. Distoma (very rare). Coccidia—Small multiple abscesses.
4. DIRECT EXTENSION from neighbouring inflammatory foci. Right empyema, peritonitis, right perinephritis, etc.
5. PYÆMIC CAUSES.
   - THROUGH PORTAL VEIN (pyogenic cocci: *B. coli communis, Amœba coli*).—Ulcers of stomach or duodenum—Appendicitis—Dysenteric ulcers—Abscess of pancreas or spleen—Operations for piles or lithotomy—Pelvic peritonitis—Septic inflammation of stump of umbilical cord—Gastro-intestinal catarrh.
   - THROUGH THE HEPATIC ARTERY.—Septic injuries of cranium—(COMMONEST). Septic inflammations of bone, especially otitis media. Ulcerative endocarditis. Septic diseases of lung.
6. TYPHOID FEVER.—Direct extension up bile-ducts. By pylephlebitis. Indirectly through a focus of suppuration.
7. TROPICAL ABSCESS.
   - GEOGRAPHICAL DISTRIBUTION.—Commonest in tropics. Mediterranean litoral. Occasional in England.
   - CLIMATE AND SEASON.—Greatest moisture, and greatest daily variation in temperature.
   - RACE.—Europeans are more subject than natives.
   - SEX AND AGE.—Men to women as 30 to 1. Age 20 to 40.

Alcohol is great predisposing cause.

Malaria may act as a predisposing cause, but tropical abscess often occurs where malaria is unknown, e.g., Chili.

Dysentery—In 75 per cent cases. *Amœba coli* found in stools and in the abscesses.

Pathology.

SINGLE.—Tropical, in 75 per cent—Gall-stones—Ascarides—Spreading from neighbouring suppuration.

MULTIPLE in all other cases ; typically the pyæmic.

MAY OPEN into : Lungs—Pleura—Pericardium—stomach—Intestine—Peritoneum—Pelvis, right kidney—Vena cava—Externally.

SITUATION.—Commonest in upper part and back of right lobe.

Symptoms.—Often latent until rupture (tropical) :

Prominence, or bulging, in hypochondrium. Redness and œdema of skin.

Dull aching Pain over liver. Sharp pain indicates perihepatitis.

Enlargement of Liver.—Swelling moves with respiration. Fluctuation is rarely felt.

Muddy Complexion.—Slight jaundice.

Auscultation.—Friction sounds over liver. Signs of pleurisy at base of right lung. Crepitations at base of right lung. Splashing sounds when abscess has burst into the pleura.

Enlargement of liver dullness, generally upwards. Stomach, colon, and right kidney pushed down. Pain in right scapula and shoulder. Cough (from irritation of phrenic or invasion of lung). *Amœba coli* may be present in sputum. Dyspnœa from encroaching on chest.

Temperature.—Hectic (with rigors) in cases of injury or pyæmia. Often normal in latent tropical abscess.

Digestive Disturbances.—Vomiting, diarrhœa, constipation, etc.

Complications caused by abscess bursting :—

Into Stomach.—Vomiting of pus.

Into Intestine, generally colon—Pus per anum.

Into Peritoneum.—Fatal peritonitis.

Into Pelvis of Right Kidney.—Very rare.

Into Inferior Vena Cava.—Fatal.

Into Gall-bladder.—Pus by bowel. May recover.

Into Pleura.—Rare—Symptoms of rapidly-formed empyema.

Into Lung.—Signs of pleurisy—Cough. Expectoration of foul, dark pus, often much blood—*Amœba coli*. Seventy per cent tropical cases in which this happens end well, but in pyæmic cases it is fatal.

Into Pericardium.—Rapidly fatal.

Externally : between or below the costal cartilages.

Nervous System.—Attacks of melancholia or hypochondriasis. Delirium and coma at end.

Abscess of the Liver, *continued.*

Diagnosis.

LATENCY.—Some cases are latent, and bursting of abscess is first sign.

SPECIAL SIGNS.—Pain in right hypochondrium—Pain in right shoulder—Muddy complexion—Jaundice (slight)—Enlarged liver —Raised temperature.

PRESENCE OF DYSENTERY, septic wounds of head, or any other focus of suppuration, make above symptoms more significant.

LOCALIZED PERIHEPATITIS may give pain like abscess, but there is no enlargement of liver, and symptoms are not so severe.

OTHER VARIETIES OF SUBPHRENIC ABSCESS (see p. 299).

ASPIRATION BY NEEDLE is only justifiable if positive result leads to immediate operation. It may show *Amœba coli*, hydatid hooklets, coccidia, actinomycosis, or thick pus like anchovy sauce.

Treatment.—Freely open after locating abscess with syringe.

If parietes are not adherent, pack sponges round, and sew parietes to liver before evacuation.

Knife of thermo-cautery is best to cut liver.

If abscess be in right lobe above margin of ribs, the thoracic wall must be incised.

If abscess communicates with pleural cavity, part of chest wall must be freely excised, and both cavities drained.

If empyema exists, but does not communicate with liver, then two separate openings are required.

If abscess burst into peritoneum, open at once, and, if adhesions, localize the mischief, then evacuation may be successful.

Prognosis.—In single or non-pyæmic cases, mortality varies 50 to 80 per cent.

HYDATID CYSTS.

Distribution.—Common in Australia and Iceland. Commoner in London than in Scotland or provinces.

SITUATION in liver.—Any part or surface may be involved. Hydatids of liver form 57 per cent of hydatid disease.

Symptoms of hydatid cyst of liver :—

HEPATIC TUMOUR.—On the superior surface extending upward, liver pushed down. On the inferior surface extending downward, liver pushed up. Fluctuation, or elasticity, with "hydatid fremitus."

PRESSURE SYMPTOMS.

Upon lung—Dyspnœa.
Upon digestive organs—Pain, vomiting, etc.
Upon blood-vessels—Œdema, ascites, piles.
Upon peritoneum—Localized peritonitis, pain.
Jaundice is rare—From pressure, or bursting into bile-duct.

RUPTURE.
Into alimentary canal—Spontaneous cure.
Into Peritoneum—Shock, collapse, urticarial rash.
Into pleura or lung—Pleurisy or pneumonia, possible expectoration of cysts.
Into pericardium—Rapidly fatal.
Into gall-bladder—Colic and jaundice.
Into pelvis of kidney, bladder, vena cava, portal vein.
Externally—Fistula.

SUPPURATION.—May result from injury, from presence of other septic focus, from pyæmia. Forms hepatic abscess with usual signs.

SPONTANEOUS CURE.—Parasite dies, fluid is absorbed, inert pultaceous material remains encapsuled. Cyst may become calcified. Cyst may be filled with bile, and then be cured.

HÆMORRHAGE into the sac may follow rupture, and may prove fatal.

COMPOSITION OF HYDATID FLUID.—Neutral reaction. Sp. gr. 1016. About 2 per cent solids—salts, proteids, and extractives. Often contains hooklets.

Diagnosis.—Slow-growing hepatic swelling, which is :—Fluid or elastic—Moves with respiration—Aspiration shows hooklets.

Diagnosis of hydatid cyst from :—

1. Solid Enlargement of Liver. In these: Solid feel. Signs of syphilis, or malignant disease. Aspiration produces no fluid.
2. Simple Cysts (very rare).—Fluid has no hooklets, and is rich in albumin.
3. Abscess.—Rapid development. Tender to pressure. Throbbing pain. Rigors and temperature.
4. Enlarged Gall-bladder.—Associated with colic and jaundice. Lateral movement possible.
5. Subphrenic Hydatid.—Entire liver pushed down. Pleural cavity encroached on. Costal arches everted.
6. Subphrenic Abscess.—Inferior border of liver displaced but unaltered in outline. Signs of inflammatory process. Aspiration produces pus or gas.
7. Cyst or Swelling of Right Kidney.—Moves slightly with respiration. Colon is in front, not below.
8. Pleural Effusion.—Dullness is more diffuse and less localized. Constitutional symptoms are more marked.
9. Aneurysm of Hepatic Artery or Aorta.—Expansile pulsation. Blowing murmur. Jaundice always when hepatic artery is involved. Pain very great.
10. Ovarian Cyst.—Pelvic connections. Line of separation from liver.
11. Hydatid Cyst of Abdominal Wall.—Liver felt moving independently.
12. Hydatid of Base of Lung.—Cough with blood-stained expectoration.

Hydatid Cysts—Diagnosis from, *continued.*

13. Phantom Tumour.
14. Local Abscess in Peritoneum.
15. Ascites.

Treatment.

ASPIRATION OR PUNCTURE.—Uncertain. Only succeeds in 50 per cent. Mortality 19 per cent.

INJECTION of iodine, perchloride, etc. Dangerous and uncertain.

INCISION, followed by sewing the sac to the parietes and packing, is the best treatment. May be carried out in one or two stages :—

Through abdomen in most cases.

Through thorax when cyst is on superior or posterior surface, or has burst into pleura.

EXCISION has been successful occasionally.

Prognosis.—Bad when suppuration or rupture occurs. When cyst has to be reached through thorax.

Mortality after incision through abdomen, 10 per cent. After incision through thorax, 29 per cent.

Other Cysts of the Liver—all rare.

SIMPLE SEROUS CYSTS.—May be large or small. Usually no symptoms. Fluid becomes solid on boiling. No hooklets. Probably lymphatic or bile mucous glands dilated.

Treat as if hydatid.

MULTILOCULAR CYSTIC DISEASE.—Often associated with cystic kidneys. Probably mucoid degeneration of bile capillaries. Commonest in old people. Generally not recognized.

No treatment.

CYSTIC ADENOMA.—Localized glandular tumour containing cysts.

DERMOID CYSTS.—Very rare.

MOVABLE LIVER.

Causes.

CONGENITAL.—Elongation of coronary and suspensory ligaments. Absence of coronary ligaments.

ACQUIRED.—Enlargement or growth of gall-bladder. Narrowing of outlet of thorax (tight lacing). Weight or traction of tumours or cysts. Laxity of abdomen, caused by many pregnancies. Fatty degeneration of suspensory ligament following peritonitis.

Symptoms.—Often associated with enteroptosis, movable kidney, or spleen. Dragging pains. Occasional jaundice. Tumour—shape of liver in abdomen, which can be replaced in right hypochondrium. Resonance in hepatic region. Difficulty in walking. Digestive derangements. Almost always in women.

Treatment.—Belt. Discontinue corsets. Possibly hepatopexy.

SOLID ENLARGEMENT OF THE LIVER.

Malformation.—A down-growing tongue of hepatic substance, usually connected with the anterior margin of the right lobe. Known as Riedel's lobe.

It is only of importance, because it may be mistaken for gall-bladder, kidney, or other tumours.

Actinomycosis.—Rarely primary. Usually secondary to an intestinal infection, or an extension from the thorax. The liver is enlarged, with bossy outline, and is associated with signs of abdominal or pleuritic chronic inflammation. It softens and breaks down into an abscess, the pus of which may give the clue.

Prognosis and treatment are hopeless.

Syphilis.

DIFFUSE HEPATITIS.—Usually seen in congenital cases. The liver becomes hard, nodular, much enlarged from an interstitial fibrosis.

GUMMATA.—Occur in acquired or congenital forms. They cause a bossy enlargement of the surface, with few or no symptoms unless the portal fissure is encroached upon.

Amyloid Disease may result from syphilis or chronic suppuration.

Angioma is the only innocent new growth which is not rare. May be congenital or acquired, and it usually grows just beneath the capsule.

CONGENITAL CASES form large masses reaching to the navel which are not encapsuled.

ACQUIRED CASES occur at about 60 as multiple, encapsuled tumours about ½ to 1 in. across.

Carcinoma, as a primary growth, may arise from the hepatic cells, or from those of the bile-ducts. The mass is single. Secondary growths are common and multiple.

Sarcoma is very rare primarily, but common secondary to bone and melanotic deposits.

SYMPTOMS OF MALIGNANT ENLARGEMENT.—Nodular enlargement which encroaches upon the thorax and abdomen. Jaundice, emaciation, cachexia, and ascites are common, if death does not occur from the primary disease.

NEOPLASMS OF GALL-BLADDER AND DUCTS.

Sarcoma, papilloma, and fibroma are very rare.

Carcinoma.

PATHOLOGY.—Generally scirrhous. Columnar-celled—from epithelial lining. Spheroidal-celled—from mucous glands. 90 per cent associated with gall-stones. Growth begins in mucous membrane, and at first is a localized growth.

Neoplasms of Gall-Bladder and Ducts—Carcinoma, *continued.*

SYMPTOMS.

IN EARLY STAGE: Very vague and indefinite.

IN ADVANCED STAGE:—

JAUNDICE in 70 per cent of cases. From impaction of calculus or growth, from invasion of ducts, from pressure of glands at portal fissure.

PAIN.—Sharp and paroxysmal, or constant and gnawing.

TUMOUR.—Hard, nodular. Sometimes suppurating.

Loss of weight. Spontaneous hæmorrhages. Gastro-intestinal disturbances.

SEX.—Women are attacked three times more often than males.

AGE.—Between 50 and 60.

DIAGNOSIS from carcinoma of liver secondary to other growth; carcinoma of pylorus or pancreas.

TREATMENT.—If diagnosed before infiltration of liver has begun, remove gall-bladder and resect adjacent part of liver.

PROGNOSIS.—Life lasts few months to two years.

Malignant Growths of Bile-ducts.—Columnar-celled carcinoma. Common bile-duct in duodenum is usual seat, but any other part may be affected.

Begins as a papillary or submucous growth. Constricts duct. Invades pancreas, duodenum, etc. Seldom larger than hazel or walnut.

No special relation to gall-stones. Both sexes equally affected.

SYMPTOMS.—Indefinite pains in area of eighth dorsal nerve. Gradual onset of jaundice with all its signs. Hæmorrhages—cutaneous and mucous. Skin becomes olive-green. Intense itching. Enlargement of liver and gall-bladder. Slow pulse. Cholæmia—drowsiness, vomiting, delirium.

DIAGNOSIS.

FROM IMPACTED GALL-STONE.—History of colic. Duration not more than few months in cancer.

FROM CANCER OF HEAD OF PANCREAS.—Almost impossible.

TREATMENT.—Cholecystenterostomy is the only possible operation in most cases. Results are bad. Hæmorrhage is so very liable to occur in jaundiced subjects.

Removal of growth is very seldom possible.

PROGNOSIS.—Seldom live more than three months after jaundice has become established.

CHAPTER XLI.

DISEASES OF THE PANCREAS.*

Diabetes.—Is caused by total removal or destruction of the pancreas by atrophy or new growth.

Is associated with fatty and fibrous degeneration of the pancreas in many cases.

Interacinar pancreatitis, with destruction of certain special cells called the islands of Langerhans, is the lesion most frequently found in pancreatic diabetes.

Injury.—Is very rare, and then usually fatal.

A blood-cyst may form in the lesser peritoneal cavity, and in this pancreatic ferments are present.

Treat by opening and emptying from in front, with ligature of bleeding vessels and drainage from behind.

PANCREATITIS.

Etiology.—The relation of the pancreatic duct to the common bile-duct, the two having a common opening into the duodenum, make any obstruction of the ampulla of Vater (the common opening) by a gall-stone likely to produce pancreatic obstruction and inflammation.

INJURY by an abdominal contusion or operation may lead to sub-acute or chronic pancreatitis.

BACTERIAL INFECTION from the intestine or bile-ducts.

Pathology.

ABSENCE OF PANCREATIC DIGESTION, as shown by: (1) The presence of undigested meat fibres in a patient not suffering from diarrhœa; (2) Abundant undigested fat in the motions, causing the pale stools.

FAT NECROSIS, i.e., a splitting up of fat into glycerin and fatty acids. The latter remain as opaque masses in the tissues, united to calcium salts. It is the result of the diffusion of the fat-splitting pancreatic ferment in the blood. It occurs chiefly in the abdominal fat, both visceral and parietal. It is common in acute and subacute pancreatitis, but rare in the chronic.

HÆMORRHAGE.—Is conspicuous and well marked in the majority of the acute cases. It occurs: (1) In the pancreas itself; (2) Into the peritoneal tissues around. It may be due to the action of the glycerin liberated by the fat-splitting process, and is often increased by co-incident jaundice.

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* Robson & Moynihan, "Diseases of the Pancreas."

Pancreatitis—Pathology, *continued.*

CRYSTALS IN THE URINE.—In many cases of obstructive and acute pancreatitis the urine yields a crop of fine yellow crystals when boiled with phenyl-hydrazine after oxidization (Cammidge).

Varieties.

1. Acute pancreatitis, ending fatally within two or three days.
2. Subacute pancreatitis, lasting several weeks.
3. Chronic or interstitial pancreatitis, lasting indefinitely.

Acute Pancreatitis.

ONSET is sudden, but may be preceded by a blow.

SEVERE PAIN AND TENDERNESS, and swelling in the epigastrium.

SHOCK is profound, the pulse small and quick, the temperature subnormal.

SEVERE VOMITING and marked constipation. The latter may yield to enemata and give place to diarrhœa.

Case presents signs and course of ACUTE PERITONITIS of an upper abdominal type, and ends fatally in about three days.

DIAGNOSIS is seldom possible from ruptured gastric or duodenal ulcer, and is often confused with acute obstruction.

PATHOLOGY.—Gangrene or extensive hæmorrhage into the pancreas, with marked fat necrosis.

TREATMENT.—Median incision establishes the diagnosis by finding (1) The swelling and hæmorrhage in the pancreatic region; (2) The fat necrosis. The organ should then be explored and drained through the left costo-vertebral angle.

Subacute Pancreatitis.

LESS SUDDEN IN ITS ONSET, and marked with much less collapse than the acute variety.

VOMITING is not so severe.

DIARRHŒA becomes prominent, blood and pus being passed in the stools.

A VERY IRREGULAR TEMPERATURE, rising to 103–105° F. in the evening.

LOSS OF FLESH very rapid.

MARKED TENDERNESS; a definite swelling or an abscess may form over the pancreas.

THE ABSCESS may track: (1) Up as a subphrenic; (2) Out as a peri-renal; (3) Down as an iliac or psoas; or (4) Forwards as a peritoneal abscess.

Case lasts for weeks or months, and may recover.

TREATMENT.—Calomel and salol by mouth. Gastric lavage, and enemata.

OPERATION FOR ABSCESS.—Exploratory from in front. Draining behind or in front, after having packed off the peritoneal cavity.

Chronic Pancreatitis.

CAUSES.—(1) Biliary catarrh or calculi in the termination of the common bile-duct, which also obstruct the duct of the pancreas; (2) Catarrh, ulcer, or sepsis in the duodenum; (3) A pre-existing acute or subacute pancreatitis.

ONSET is usually gradual, but it may be sudden, with pain and jaundice.

PAIN may be both continuous and paroxysmal, and in both cases is in the epigastrium above the navel.

JAUNDICE is well marked, and deepens with each paroxysm of pain.

ASTHENIA, with loss of flesh and digestive disturbance, e.g., dyspepsia and vomiting, are well marked.

STOOLS are copious, loose, light-coloured, offensive, and contain free fat.

HECTIC FEVER with rigors is not infrequent.

THE HEAD OF THE PANCREAS can be felt as a hard tumour, and often the gall-bladder is distended.

VARIETIES.

1. Syphilitic, found in the congenital disease in infants.
2. Interlobular. Affects the tissue between the lobules, and the islands of Langerhans escape. It is the common form arising from duct infection, and produces no glycosuria.
3. Interacinar. The inflammatory fibrous tissue penetrates the lobules and invades the islands of Langerhans. It is associated with diabetes.

ANATOMY.—Suppurative catarrh of the pancreatic ducts. Interstitial inflammation of the gland, with swelling at first and contraction later. Adhesions of duodenum, pylorus, and gall-bladder, with distention of the latter.

DIAGNOSIS.—Is usually confused with chronic gall-stone cases, and only recognized during the operation. The tenderness in the mid-line, and tumour, may make it clear.

CANCER OF THE HEAD OF THE PANCREAS is often indistinguishable from this disease. The onset is in older people, is more painless, and the course more rapid.

COURSE.—May last for years unless glycosuria supervenes.

TREATMENT.—Drainage by the CHOLECYSTOSTOMY operation. If a calculus be in the ampulla of Vater, it should be removed through the duodenum.

Pancreatic Calculus.—Very rare. Composed chiefly of carbonates. Cause colic, and are associated with diarrhœa, and often diabetes.

Pancreatic Cysts.

CLASSIFICATION.—(1) Retention; (2) Proliferation; (3) Hydatid; (4) Congenital; (5) Hæmorrhagic; (6) Pseudo-cysts.

Pancreatitic Cysts—Classification, *continued.*

1. Retention Cysts.—Caused by a calculus, interstitial fibrosis, pressure of a tumour of the duodenum, or kinking. Form the majority of simple cysts.
2. Proliferation Cysts.—Either adenomata, like ovarian tumours, or carcinomata.
3. Hydatid Cysts.—Very rare.
4. Hæmorrhagic.—May be left after the absorption of blood from a cavity in or round the pancreas.
5. Congenital Cystic disease, similar to that of the kidneys and liver.
6. Pseudo-cysts.—Lying in front of the pancreas, often following trauma. Probably are simply formed by the filling of the lesser peritoneal sac with fluid exuded by injury of the pancreas.

ANATOMY.—Usually in the body of the gland and often of large size, holding fifteen to twenty litres. The inner wall is smooth, but in proliferation cysts there are sometimes papillomata. Dense adhesions are the rule.

Fluid is dark, viscid, and mucoid, and may contain proteolytic, emulsifying and amylolytic ferments.

SYMPTOMS.—Epigastric pain. Dyspepsia, vomiting, and rapid loss of flesh. Alterations in the fæces or urine (fat or sugar) are exceptional. Absence of pancreatic secretion in the intestine is shown by Sahli's sign, viz., the fact that salol is not decomposed into salicylic and carbolic acids, whose presence in normal cases can be detected in the urine.

SIGNS.—An epigastric elastic swelling rather to the left of the mid-line. It is usually behind the stomach and transverse colon. It may take four different courses in its growth :—

1. Between the stomach above and the colon below, both lying in front of it. This is much the commonest.
2. Above the stomach, behind the gastrohepatic omentum.
3. Into the layers of the transverse mesocolon.
4. Below the transverse colon.

When it becomes large it fills the whole abdomen. It is usually fixed, and moves very little with respiration.

DIAGNOSIS.—From cysts of the left kidney, liver, spleen, mesentery, omentum, and ovary.

TREATMENT.

Evacuation and Drainage.—Median incision. Opening the cyst between the stomach and transverse colon, stitching it to the parietes, and draining.

Extirpation is rarely possible.

Carcinoma.—May be scirrhous, encephaloid, columnar, or colloid. Usually in the head of the gland. It spreads thence into the bile-duct, duodenum, colon, ureter, portal vein.

SYMPTOMS.—Pain is gradual in onset, but usually severe. Jaundice with enlargement of the gall-bladder is chronic. Very deep, and often accompanied by enlargement of the liver. Tumour is only felt in about a quarter of the cases. Rapid wasting, vomiting, copious foul stools containing undigested fat and muscle fibre.

COMPLICATIONS.—In addition to the usual (1) Biliary obstruction, there may be: (2) Pyloric obstruction; or (3) Ascites from pressure on the vena cava and portal vein.

DIAGNOSIS.—FROM GALL-STONES, by the painless onset of jaundice, the enlargement of the gall-bladder, and the rapid emaciation.

Other New Growths are adenoma and sarcoma. Both very rare.

CHAPTER XLII.

AFFECTIONS OF THE KIDNEYS AND URETERS.*

CONGENITAL ABNORMALITIES.

Misplacements.—The kidney may be situated over the sacro-iliac joint, in the pelvis or near the internal abdominal ring, whither it is probably dragged by the descent of the testis. This extreme displacement, which is very rare, may cause pain especially during menstruation or pregnancy.

Malformation.—Division into two or many lobes, such as is normal in many animals, is comparatively common but of no clinical significance.

DOUBLE URETER, which may affect only the upper part or the whole length to the bladder, is fairly common.

SUPERNUMERARY VESSELS are common. The accessory renal artery usually comes off separately from the aorta below the main renal artery, and it may form a band over which the ureter becomes kinked, thus bringing about hydronephrosis.

Number.

ABSENCE OF ONE KIDNEY is excessively rare, probably less than 1 in 5000 cases.

CONGENITAL ATROPHY OF ONE KIDNEY is commoner than the above, and probably accounts for many cases which are included in that category. The kidney is represented by a fibrous mass, the ureter by a slender cord, and its vesical orifice by a dimple. In both this and the last the opposite kidney is hypertrophied to nearly double the normal size.

FUSION OF THE TWO KIDNEYS INTO A SINGLE MASS.—This is the most important of all the serious forms of congenital abnormality. It usually forms a horse-shoe viscus, the two kidneys being united at their lower pole and generally being lower than normal. They may form a disc or S-shaped organ in one lumbar region or in the pelvis. Unlike the conditions when one kidney is atrophied or absent, it gives no evidence by cystoscopic examination, as the ureteric orifices are normal.

MOVABLE KIDNEY.

Degrees of Mobility.

1. MOVABLE BEHIND THE PERITONEUM.—"Cinder-sifting mobility." Either in or with its fatty capsule.

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* Henry Morris, "Surgical Diseases of the Kidneys and Ureters."

2. FLOATING FORWARDS as well as downwards, pushing the peritoneum before it.
3. FLOATING WITH A DISTINCT MESONEPHROS or peritoneal pedicle.

Etiology.—Women form 80 per cent of cases. Right kidney affected twelve times as often as left. Age at which symptoms are most marked, thirty to forty-five.

OFTEN FOLLOWS: Repeated pregnancy. Rapid emaciation which deprives the kidney of some of its fat capsule. Tight lacing, pushing down the liver and kidney. Fluxional hyperæmia associated with menstruation. Blow on the loin, or violent fall on the feet. Any enlargement of the kidney.

OFTEN ASSOCIATED WITH: Dilatation of the stomach—Enteroptosis—Melancholia.

Anatomy.—Scarcity or absence of perinephric fat. Laxity of peritoneum. Kidney is large, soft, and flabby. Renal vessels elongated.

Complications.—Dilated pelvis and hydronephrosis. Frequent kinking of the ureter. Pyelitis. Calculus, tumour, or other disease. Lower pole is tilted forward and inward.

Physical Signs.—Bimanual palpation can grasp the lower pole of the kidney. Abdominal tumour is formed when the kidney "floats." It is generally just at the side of the umbilicus. Abnormal resonance in the loin.

Symptoms.

PAIN.—Uncomfortable sense of weight or movement in the loin. Dragging pain in the abdomen and back, worse during the menstrual periods. Occasional crises of acute agony if the pedicle becomes twisted.

GASTRO-INTESTINAL SYMPTOMS.—Dyspepsia and dilatation of the stomach, possibly due to a kinking of the duodenum. Colic with constipation, or rarely diarrhœa, from a dragging on the colon. Violent gastric crises of colic and vomiting from a dragging on the renal pedicle. Transient jaundice.

URINARY SYMPTOMS.—Slight albuminuria, pyuria, or hæmaturia, from pyelitis or congestion. Polyuria, with ardent desire to micturate. Pain and scanty micturition, alternating with cessation of pain and polyuria, points to kinking of the ureter and hydronephrosis. Acute pain, passage of albumin, blood, and casts, point to torsion of the renal pedicle.

NERVE SYMPTOMS.—Hysteria, neurasthenia, melancholia, and hypochondriasis. Often worse during menstruation.

Diagnosis.

FROM OTHER KIDNEY DISEASES, e.g., calculus, tubercle, or new growth.

In all these some distinct feature, e.g., crystals, bacilli, or hæmaturia, is present, and the course of the disease is more definite and rapid.

Movable Kidney—Diagnosis, *continued.*

FROM OTHER FORMS OF CHRONIC ABDOMINAL PAIN.—Ovaritis or prolapsed ovaries. Uterine displacements. Recurrent appendicitis. Colic and constipation.

FROM OTHER ABDOMINAL SWELLINGS.—Abnormal right lobe of the liver. Gall-bladder distention or growth. Abnormal spleen. Growths of the cæcum. Ovarian tumour.

The fact that the kidney can be replaced in the loin, and the sickening pain felt on pressure on the kidney, are the main facts which prove a swelling to be displaced kidney.

Prognosis.

PROSPECT OF CURE.—After inflammatory attacks. If the patient becomes fat. Sometimes after pregnancy.

PROSPECT OF BECOMING WORSE.—When associated with enteroptosis or hydronephrosis.

PROSPECT OF DANGER.—When associated with occurrence of renal crises; hydronephrosis or pyonephrosis; increasing constipation or appendicitis.

Treatment.

PALLIATIVE.—Indicated in: (1) Mild cases; (2) Cases associated with hysteria or hypochondriasis; (3) General enteroptosis.

CONSISTS OF: Rest, fattening diet. Belt with pad over kidney region: must be applied when the kidney is replaced.

RADICAL.

1. NEPHROPEXY.—When symptoms are severe and not relieved by rest and belts. When hydronephrosis is present. When renal crises occur.

   Results: 1–2 per cent die; 90 per cent are cured of the pain; 50 per cent are cured of gastro-intestinal symptoms.

2. NEPHRECTOMY.—Only when the kidney is disorganized by hydronephrosis or other disease.

INJURIES OF THE KIDNEY.

Sub-parietal Injuries.

Causes.—Direct violence. Crushes. Indirect violence. Impact of ribs, and muscular violence.

Occurs.—Most commonly in men, and on the right side.

Anatomy.—Any of the following lesions may occur: Fatty capsule torn—perinephric hæmatoma. Peritoneum torn—especially in children. Subcapsular hæmorrhage. Rupture of the parenchyma—hæmatoma. Rupture of pelvis—bleeding and extravasation. Rupture of vessels. Thrombosis of vessels. Total pulping of kidney. Tearing of the kidney from its hilum. Necrosis or abscess of the kidney.

Symptoms.

COLLAPSE.—Long and profound. Often delayed when it is due to hæmorrhage. Vomiting.

ECCHYMOSIS.—In kidney region. Round the external abdominal ring and genitals.

PAIN.—In loin, radiating to testis and thigh. Pain and retraction of testis when clot is in the ureter.

TUMOUR.—In kidney region. Painful and ill-defined.

HÆMATURIA.—Commonest and most characteristic sign.

IT RESULTS FROM: (1) Rupture of the kidney; (2) Simple contusion or congestion; (3) Embolus or thrombosis; (4) Late inflammation; (5) Pre-existing cause, e.g., stone.

IT MAY BE ABSENT in ruptured kidney: (1) When rupture is superficial; (2) Vessels thrombosed; (3) Ureter plugged; (4) Kidney torn from the ureter.

CHARACTERS.—Profuse. Lasts many days, but may be intermittent. May have clot casts of the ureter.

MICTURITION.—Urgent desire for micturition. Pain and difficulty in the act. Pus and casts in the urine from late nephritis.

OLIGURIA OR ANURIA.—From shock. Injury of solitary kidney. Injury of both kidneys.

RETENTION OF URINE (rare).—Blood-clot in the bladder. Paralysis of abdominal muscles.

PERITONITIS.—When the peritoneum has been torn.

Results of severe injuries: (1) DEATH from shock or hæmorrhage; (2) PERITONITIS; (3) INFLAMMATION and suppuration; (4) OCCLUSION of ureters, with hydronephrosis, pyonephrosis, or atrophy of the kidney; (5) EXTRAVASATION of urine; (6) TRAUMATIC NEPHRITIS.

Treatment.

FOR SHOCK AND IN MAJORITY OF CASES.—Morphia and Ergot. Purgation. Leiter's tubes.

FOR ACTIVE ARTERIAL HÆMORRHAGE.—Exploratory incision—Pack rupture with gauze. Wash out bladder if clots cannot pass. Cystotomy if bladder is full of clot.

IN SEVERE CASES.—Clamp pedicle. Remove kidney later if the shock is severe.

IN PERITONEAL EFFUSION.—Continue loin opening in front.

LUMBAR HÆMATOMA.—Open and drain.

Incised and Penetrating Wounds.

atomy and Symptoms the same as above, with addition of: —External hæmorrhage—Ecape of urine.

ers.—Involvement of vessels or hilum. Wound of the Septic processes.

Injuries of the Kidney—Incised and Penetrating Wounds, *continued.*

Treatment.

WOUND.—Enlarge a small wound. Pack and drain a large one. Locate and remove any foreign body.

HÆMORRHAGE.—Expose the kidney. Sew, pack, or remove, according to the degree of gravity.

PERINEPHRITIS. PERINEPHRIC ABSCESS.

Varieties.—Sclerosing—Lipomatous—Purulent.

Etiology.

PRIMARY, i.e., UNCONNECTED WITH KIDNEY.—Traumatic—Sudden chill—Sequela of exanthemata.

SECONDARY TO SOME KIDNEY LESION.—Pyelitis—Pyelonephritis—Calculus—Cancer or tubercle—Disease of urethra, bladder, or prostate.

SECONDARY TO OTHER DISEASED ORGAN.—Pelvic inflammation. Disease of colon, appendix, gall-bladder, testis, urethra, rectum, uterus, liver, vertebræ.

Symptoms.

1. WITHOUT SUPPURATION.
   - PAIN in loin.
   - STIFF SPINE inclined to tender side.
   - STIFFNESS IN WALKING and inclination to one side.
   - FLEXION OF THIGH and LORDOSIS. Complete extension of thigh impossible. Flexion, abduction, and adduction can take place. Can stand on bad leg if body is bent over to bad side. Sometimes pain in knee.
   - TEMPERATURE 103°–104° F.
   - ABSENCE OF SIGNS OF DISEASE OF HIP JOINT.
2. WITH SUPPURATION.
   - CONSTITUTIONAL SYMPTOMS.—Temperature, etc.
   - OBSTINATE CONSTIPATION.
   - PAIN deep-seated, throbbing, radiating, and variable. Increased by pressure.
   - INCREASED RESISTANCE and weight in loin. Dullness to percussion, fluctuation. Redness and œdema of flank.
   - RETRACTION OF TESTIS on affected side.
   - ŒDEMA OF FOOT AND LEG on affected side.
   - Occasional anæsthesia or paralysis on affected side.
   - URINE.—Exceptionally contains blood or pus.

Pathology.

THE ABSCESS varies from small to very large. Often gangrenous walls.

PUS IS FÆCAL, or with fæcal odour. Softening and inflammation of kidney. Pus is usually behind kidney. May bulge peritoneum forward. Often dense adhesion between capsule and adipose tissue.

PUS MAY BURST into: Colon, small intestine or stomach, pleural sac or lung (commonest); ureter, peritoneum, psoas, and thence to groin or buttock, loin.

THE NON-SUPPURATIVE VARIETIES result in dense fibrous adhesions or a fibro-fatty mass round the kidney, which is compressed and itself undergoes fatty degeneration.

Prognosis.—Generally grave—30 per cent die. If opened early, good. If abscess bursts into lung, colon, or ureter, there is still a chance. If abscess bursts into pleura or peritoneum, very bad.

Diagnosis from :—

LUMBAGO.—No fever or swelling; bilateral.
NEPHRALGIA.—Periodic, local swelling absent. Hysteria.
ORGANIC DISEASE.—Better outline. No local heat or œdema.
SPINAL DISEASE.—No inclination to one side. Angular curvature.
MORBUS COXÆ.—Local signs of hip joint.
SACRO-ILIAC DISEASE.—Local signs of sacro-iliac joint.
PSOAS ABSCESS.—Nearer mid-line. Spinal symptoms.
BLOOD EXTRAVASATION.—Relation to injury. Absence of fever.
APPENDICITIS.—Symptoms begin in iliac fossa.
FÆCAL ACCUMULATION.—Splenic and hepatic tumours.
Typhoid fever, empyema, pneumonia.

Diagnosis of Position.

UPPER TRACT causes chest symptoms, e.g., pleurisy.
MIDDLE TRACT causes pain in groin, scrotum, with albuminuria.
LOWER TRACT causes pain in hip, thigh, and knee.

Treatment.—Hot fomentations. Ointment of iodide of lead, etc. Morphia. Aperients, etc.

EARLY AND FREE INCISION, with drainage.

INTERSTITIAL NEPHRITIS.

(Pyelonephritis without suppuration).

Etiology.—(1) All conditions which give rise to obstruction; (2) Traumatism; (3) Drugs—turpentine, cantharides (?).

Symptoms.

1. CHRONIC FORM.—Arising from obstruction.

INSIDIOUS ONSET; generally no rigor or chill.

GENERAL MALAISE.—Small and rapid pulse. Drowsiness. Loss of appetite. Flatulence. Rapid emaciation. Thirst, nausea, and vomiting.

HECTIC.—Evening temperature 100°–102° F. For days and weeks, with variations. Profuse sweats.

URINE.—Condition often obscured by cystitis, etc. Abundant. Low specific gravity. Trace of albumin. Pus from pyelitis.

REMISSIONS and relapses.

Interstitial Nephritis—Symptoms, *continued.*

2. ACUTE TRAUMATIC NEPHRITIS.
   - BEGINS with well-marked rigor.
   - HECTIC.—Well marked.
   - PAIN in loin generally one-sided. Very variable—acute or dull. Circumscribed or diffuse.
   - URINE.—Often suppressed. Blood. Red sandy deposit. Albumin and trace of pus.
   - TYPHOID STATE in severe infections.

Diagnosis.

CHRONIC FORM:—From any form of chronic blood-poisoning.
ACUTE FORM:—From septicæmia or typhoid.

Prognosis.—Depends on cause of obstruction. Suppression is very dangerous.

Pathology.—Thickened granular capsule. Excessive and adherent perinephric fat. Pelvis generally dilated or inflamed. Increase of interstitial connective tissue, consequent distortion of tubules. Often associated with resulting calculus. Old cases produce "cicatricial contracted kidney."

Treatment.

PROPHYLACTIC.—Overcome causes of retention. Careful instrumentation. Washing out decomposed urine from bladder.

PALLIATIVE.—Rest. Warmth. Aperients. Hot baths. Packs. Cupping and hot applications to loins. Morphia gr. $\frac{1}{6}$, atropine gr. $\frac{1}{100}$, for suppression. Quinine gr. v–x with opium ♏v–x, for hectic. Ergot for polyuria.

SUPPURATION OF THE KIDNEY.

Methods of Infection.—(1) Through circulation; (2) From lower urinary tract; (3) From spread of neighbouring suppuration; (4) From a combination of above.

Varieties.

1. DIFFUSE.—Suppurative nephritis from the circulation or ascending pyelitis—Suppurative pyelonephritis—Suppurative pyonephrosis.
2. CIRCUMSCRIBED.—Renal abscess.

Bacteria.—Chiefly *Coli communis.* Streptococci and staphylococci.

Causes.

OBSTRUCTIVE.—Phimosis—Gonorrhœa—Stricture—Stone in bladder—Prostate—Bladder paralysis—Cystitis—Malignant disease—Pregnancy—Tumours—Pelvic inflammation.

GENERAL.—Diphtheria—Typhoid—Osteomyelitis—Infective endocarditis—Exanthemata—Pyæmia—Erysipelas.

DRUGS.—Turpentine and cantharides.

TRAUMATISM and calculi.

Symptoms.

ACUTE (rare).—Typhoid state. High fever, delirium, coma. Rigors.

CHRONIC.

- TEMPERATURE.—Continuously high, or alternating periods of fever with remissions of several days.
- DIGESTIVE ORGANS.—Dry mouth, cracked tongue, etc. Vomiting and flatulence. Constipation, occasional diarrhœa.
- URINE.—Large quantity, low specific gravity. Small quantity of urea. Often complicated with pus or mucus from cystitis (alkaline). Often pus in acid urine (pyelitis). Little or no albumin.
- PROFUSE SWEATING. Rigors. Clammy skin.
- PAINFUL SPASMS of neck of bladder.
- DROWSY, or later comatose.
- PAIN IN LOINS and down ureters is often present, but often absent.

Diagnosis from :—

OTHER CONDITIONS OF LOWER URINARY ORGANS.—By fever and digestive disturbance.

ACUTE BRIGHT'S DISEASE.—By absence of coma, convulsions, or œdema. Abundant urine.

PYÆMIA.—Presents secondary abscesses and more regular sweats and rigors.

SEPTICÆMIA.—Is more acute, and mental symptoms become more rapidly marked.

TYPHOID.—Widal's reaction. General course, etc.

Prognosis.—Fatal in two to four weeks, unless cause of obstruction can be removed.

Pathology.—Increase in size and in the amount of connective tissue. Adhesions to fatty capsule. Numerous foci of suppuration. Radiating yellow streaks of suppuration. Dilated pelvis, with thickened mucous membrane. Infiltration, with leucocytes and pus cells in the intertubular tissue. Proliferation of epithelium, with swelling and necrosis. Coli bacilli, chiefly in tubules. Cocci, mostly in lymphatics.

Treatment.—Chiefly prophylactic, e.g., irrigation of bladder, diet, rest, etc.

HYDRONEPHROSIS.

Causes.—Some form of gradual or intermittent obstruction.

CONGENITAL.—Phimosis. Abnormalities of ureter. Torsion of penis. Abnormalities of the renal vessels.

ACQUIRED.—Urethral stricture. Prostatic enlargement. Vesical stone or new growths. Vesical systole (sustained). Pelvic tumours or displacements. Bands or adhesions of peritonitis. Ureteral stenosis from ureteritis. Ureteral calculus, valve, or pocket. Kinking of ureter from mobility. Traumatic obstruction.

Hydronephrosis, *continued.*

Varieties.—(1) Thin-walled cyst—atrophy of parenchyma; (2) Hypertrophied parenchyma round dilated calyces.

Pathology.—Different degrees of dilatation of pelvis and calyces, with varying atrophy of parenchyma. Fluid consists of water and sodium chloride. Cells, phosphates, albumin frequent. Cholesterin occasional.

Symptoms.—Twice as common in women as in men. There are two clinical groups of cases:—

1. THOSE OF SLIGHT DEGREE, where there is: Little or no atrophy of renal parenchyma—No palpable tumour.
   SYMPTOMS.—Occasional attacks of pain in the loin and scanty micturition. Relieved by alternating polyuria.
2. THOSE OF ADVANCED DEGREE, where there are:—
   Atrophy of the renal parenchyma.
   LARGE PALPABLE TUMOUR.—Dull, large, with all the characters of a renal tumour. Fluctuates, often lobulated. May contain several gallons of fluid.
   PAIN.—Often absent. Occurs in back and along the ureter. Very severe if rupture occurs.
   INTERMITTENCE.—Pain, tumour, and scanty urine alternate with ease, no tumour, and polyuria.
   ANURIA OR URÆMIA occurs in the rare cases where the disease is bilateral.

Diagnosis from: Renal abscess—Perinephric abscess—Perinephric extravasation—Pyonephrosis—Hydatid of kidney—Hydatid of liver—Ovarian cyst—Ascites.

Treatment.—None required in small cases with few symptoms.
TAPPING.—For left kidney: spot anterior to last intercostal space. For right kidney: spot mid between last rib and iliac crest, and 2½ in. behind anterior superior spine.
NEPHROPEXY in cases resulting from mobility.
NEPHROTOMY.—Remove ureteral obstruction, or establish lumbar drain, or remove kidney, either primarily or secondarily.

PYONEPHROSIS.

Either (1) Sepsis added to hydronephrosis; or (2) Septic process and dilatation together; or (3) Septic process followed by dilatation.

Etiology.—All causes of hydronephrosis, with sepsis added. Especially calculous pyelitis. Cancer of uterus or other pelvic organ. Movable kidney. Pregnancy. Almost always on right side.

Anatomy.—Glandular structure may be destroyed. Many-chambered sac. Perinephritis, fatty, sclerosing, or suppurating. Septa between loculi consist of sclerosed parenchyma. Loculi may be separate or communicate with one another, or with a central cavity. Cavity is often shut off from the ureter.

ADHESIONS form, and FISTULÆ may follow, into the loin, colon, stomach, peritoneum, diaphragm (on left).

Contents.—Pus and urine in different proportions. Cheesy material. Calculi either primary or secondary.

Symptoms.—Generally preceded by those of cystitis or obstruction. In acute cases, rigors, diarrhœa, and sickness. In chronic cases, hectic condition. Emaciation and prostration.

TUMOUR.—Often extends upwards, covered by ribs so as to be impalpable. Sometimes is lobulated and fluctuating. Sometimes varies in size.

PAIN.—Dull ache, with acute exacerbations. Tenderness most marked in front.

URINE.—Varying amount of pus, little blood. Intermittent pyuria. Often masked by cystitis.

Diagnosis.—Especially from hydronephrosis, perinephric abscess, pyelonephritis, tubercle of kidney.

Prognosis depends on condition of other kidney and curability of cause.

Treatment.

1. REMOVE CAUSE, e.g., Stricture, prostatic enlargement, stone in bladder, etc.
2. TREAT CYSTITIS.
3. DILUENT DRINKS. Milk diet. Urotropin, etc. Fomentations.
4. OPERATION.—When diagnosis is plain.

OPERATIVE TREATMENT.

1. Nephrotomy, with free drainage.
2. Remove calculus.
3. Break down septa between loculi.
4. Nephrectomy in cases where nephrotomy has failed, or when kidney is hopelessly disorganized.

RENAL TUBERCULOSIS.

Etiology.

CHANNELS OF INFECTION.—Blood stream—Lymphatics—Ureter—Contiguity (rare).

PREDISPOSING CAUSES.—General debility—Local debility (retention of urine)—Traumatism.

AGE.—Miliary, among children (under ten). Caseous, among young adults.

BOTH KIDNEYS generally affected in miliary tubercle. In caseous tubercle, in about half the cases only one is diseased.

Renal Tuberculosis, *continued.*

Pathology and Morbid Anatomy.

ACUTE MILIARY.—Grey granulations in course of blood-vessels, chiefly in cortex.

INFILTRATING OR CASEOUS.—In ascending tuberculosis the pyramids are most involved, and large vomicæ open into the pelvis.

IN DESCENDING TYPE, where infection is by the blood-vessels, the caseous masses are most developed in the cortex. Openings of vomicæ into pelvis are small. Ureter and pelvis comparatively healthy.

IN VERY CHRONIC CASES there is only an "intertubular cirrhosis."

- URETER is often blocked with cheesy débris, with resulting hydro- or pyonephrosis.
- CAVITY OF PELVIS AND URETER are often ulcerated, and ulceration extends into parenchyma, forming large, irregular cavities.
- SECONDARY INFECTION by the colon bacillus and septic cocci occurs later.

Complications.—Tuberculous infection of: (1) Opposite kidney in 50 per cent of cases; (2) Vesiculæ seminales, vas, epididymis; (3) Bladder and other organs. Varying degrees of perinephritis, with great thickening.

Symptoms.

PAIN.—Slight at first—Severe later—Attacks of colic from blocked ureter—Some tenderness on palpation.

TUMOUR.—Moderate size, often due to perinephritis—Ballottement well marked—May be lobulated.

URINE.—Polyuria—Acid—Pyuria and hæmaturia—Urea and phosphates diminished—Great variations due to blocking ureter.

PYURIA.—Dirty grey pus in small quantity—Mixed with caseous masses and phosphatic débris.

HÆMATURIA.—Very constant symptom—Slight in amount—Sometimes clots moulded in ureter.

BACILLI.—Tubercle bacillus in small numbers and difficult of detection. Septic organisms become abundant late in the disease. Absence of other organisms in acid purulent urine points to tubercle.

CYSTOSCOPY.—Ureteral orifice is red, with pouting, swollen lips, or retracted. Methylene blue given as a hypodermic is not excreted, or excreted very slowly, by the diseased kidney.

PER RECTUM OR VAGINAM.—The thickened ureter may sometimes be felt.

ALBUMINURIA.—Always present when pelvis is diseased.

POLYURIA.—Very marked, especially at night.

DYSURIA.—Pain in neck of bladder. The earliest symptoms. Sometimes due to phosphatic débris in bladder.

CONSTITUTIONAL.—Evening temperature raised to 100–101° F.; if secondary sepsis has occurred, to 103–104° F. Loss of flesh. Anæmia. Rigors and sweats in last stage.

Diagnosis.

FROM PYELONEPHRITIS.—History of case. Thickening of ureter. Presence of tubercle bacilli.

FROM CALCULUS OF KIDNEY.—Constitutional condition. Vesical irritation. Hæmaturia occurs during rest, and is slight. Tubercle bacilli, especially by inoculation experiments.

FROM NEW GROWTHS.—Hæmaturia is much less abundant. Constant pyuria. Hectic.

Diagnosis of which Kidney.—Side of tenderness, pain, or tumour. If there is intermittent pyuria it is evidence that one kidney is healthy. Cystoscope and ureteral catheter. Methylene blue test.

Prognosis.—May last five years. Secondary pyogenic infection is the gravest complication. Occasionally undergoes spontaneous cure. Operative prognosis is about 20 per cent mortality. Ultimate result in successful cases is very good.

Treatment.

GENERAL.—Diet, climate, etc.

OPERATIVE TREATMENT.

1. Nephrectomy.—Where opposite kidney is sound. Operation of emergency for hæmorrhage, colic, pyonephrosis: Useless when advanced tubercle exists elsewhere.
2. Nephro-ureterectomy.—When ureter is diseased.
3. Nephrotomy.—Where pyonephrosis is chief symptom. Where other kidney is uncertain.
4. Partial Nephrectomy.—When disease is limited to a part of the kidney.

RENAL CALCULUS.

Structure.

PRIMARY.—	Uric acid Urates of soda or ammonia Oxalate of lime	In acid urine.
	Phosphate of lime Carbonate of lime	In alkaline urine.
	Cystin Xanthin Indigo	In acid urine, but all rare.

Renal Calculus—Structure, ***continued.***

SECONDARY.—(To septic processes).
Mixed phosphates
Phosphates of ammonia and magnesia
Carbonate of lime
From alkaline urine.

Large Calculi consist of secondary deposits on primary nuclei.

Nucleus.—Urate of ammonia when formed in infancy. Uric acid, adults. Oxalate of lime, after forty. Bacteria—only exceptionally.

Number.—Oxalate generally single. May be up to 200.

Proportion of Different Varieties.—Uric acid is 75 per cent of all calculi. Oxalate of lime is the majority of those removed, because they cause more suffering.

APPEARANCE AND CONSISTENCE.
Uric acid—hard, smooth, fawn red.
Urates—light yellow, soft, friable.
Oxalate—hard, rough, dark.
Phosphates—chalky, mortary.
Carbonate of lime—round, white, hard.
Cystin—soft, crystalline, yellowish green.
Xanthin—smooth, hard, cinnamon red.

SITUATION.—Pelvis, calyces, ureteral orifice, or parenchyma.

Etiology.—In both kidneys in 50 per cent of all cases.

Age.—Found at all ages. Clinically very rare below ten years; common between twenty and fifty.

Sex.—Slight preponderance of males.

Heredity.—In uric acid stone there is generally a well-marked family history.

Side.—Right and left equally affected.

Habits.—Sedentary life and rich diet.

Water.—Lime in drinking water.

Climate.—More frequent in hot countries, because evaporation and sweating concentrate the urine.

Injury.—May give rise to calculus round a blood-clot, or first give symptoms by moving a calculus that has been present.

Pathogenesis.

URIC ACID.—Especially before tenth and after fortieth year. Derived from nuclein. Acidity of urine. Rich diet, little exercise. Hepatic congestion. Gout. Pernicious anæmia, leukæmia.

URATES.—Especially in children. Concentrated urine as in febrile conditions. Dyspepsia.

OXALATE OF LIME.—Commoner in men than women. Nervous, irritable temperament. Studious sedentary habits. Malaria. Rhubarb, gooseberries, tomatoes, sorrel. Poor vegetable diet. Excess of acid. Formed often from uric acid. Much lime in water is excreted as oxalate.

PHOSPHATE OF LIME AND PHOSPHATE OF MAGNESIUM.—Due to presence of fixed alkali in urine. Excess of alkaline food. Excess of lime in food. Granular kidney. Phthisis. Dyspepsia.

AMMONIO-MAGNESIC PHOSPHATE.—Due to inflammation producing ammoniacal urine.

CARBONATE OF LIME.—Same conditions as phosphates. Especially associated with pus.

CYSTIN.—Contains sulphur. Runs in families.

AGGLUTINATION OF CRYSTALS to form a stone can only take place in presence of a colloid substance, e.g., colouring matters of urine, mucus, pus, or albumin.

Pathology of Calculous Kidney.

ASEPTIC CHANGES.

NEPHRITIS.—Desquamative, interstitial.

Hypertrophy first. Atrophy later: (1) From sclerosis; (2) From pressure of hydronephrosis. Hydronephrosis. Partial hydronephrosis. Increase and induration of fibro-fatty tissue. Kidney converted into lipomatous mass.

SEPTIC CHANGES.—Suppurative pyelonephritis, with secondary calculi. Pyonephrosis. Perinephritis and perinephric abscess. External or internal fistula.

CHANGES DUE TO PASSAGE DOWN URETER.—Impaction, ulceration, etc.

Symptoms.

COLIC.

CAUSED BY: Passage of stone to bladder—symptoms will cease unless second stone forms or only a fragment of first is passed. Impaction of stone in pelvis. Passage of blood-clot or mucopus.

PAIN shoots down leg, testicle, groin, or bladder. Is paroxysmal.

Testicle is retracted and becomes tender.

RIGOR, VOMITING, perspiration, collapse.

URINE is scanty, blood-stained. Frequent micturition, often scalding.

Attack ends suddenly.

Stone may be passed soon or impacted in urethra.

PAIN OF A CONSTANT CHARACTER.—In loin, abdomen, in course of ureter, testicle, thigh, inner side of leg or foot. Dull ache up to lancinating pain. Much worse after movement. Often worse at menstrual periods. Uric acid—least pain. Oxalate—acute pain. Phosphates—great and unremitting pain.

REFLEX PAINS: In opposite kidney. In bladder—frequent and painful micturition. In uterus and ovary (especially of same side). In testicle. In gastro-intestinal tract—colic, vomiting, and nausea.

Renal Calculus—Symptoms, *continued.*

HÆMATURIA.—Almost always present (microscopically if not otherwise). Often associated with pain or colic—generally profuse for two or three days after colic ceases. Caused or increased by movement. Abated or stopped by rest.

PYURIA.—Generally only slight or microscopical. Urine is acid, and pus deposits readily.

URINE.—Hyaline casts—Slight albumin—Pus, blood, crystals.

PHYSICAL EXAMINATION.—Can be felt in thin subjects very rarely. Tenderness and pricking pain on bimanual examination. Greater resistance of muscles on affected side.

URETERAL CATHETER and CYSTOSCOPE show: Œdema of one ureteral orifice. Difference of urine on two sides.

RADIOGRAPHY.—Oxalate of lime give best shadows. Phosphates give dark shadows. Pure uric acid and urates give faint shadows.

Diagnosis.

I. FROM OTHER RENAL DISEASES.

1. RENAL TUBERCULOSIS.—In this there are: Pain and hæmaturia, not influenced by rest. Colic only occurring when mucopus is passed—Tubercle bacilli in the urine, or other tuberculous lesions.
2. RENAL TUMOURS.—In these: Tumour rapidly grows. Hæmaturia is much more copious. Pain is more constant and less colicky.
3. MOVABLE KIDNEY.—In this: The kidney mobility can be felt. There is absence of crystals in the urine. The two conditions may co-exist.
4. URETERITIS OR KINKING of valve or the ureter.—In these: Intermittent hydronephrosis. Absence of crystals. The two conditions often co-exist.
5. LITHIASIS.—In this: Hæmaturia, pyuria, or albuminuria are rare and scanty. The symptoms rapidly disappear under suitable treatment with alkalies, piperazine, etc.
6. NEPHRITIS.—In this: Urine is of low specific gravity. Casts are frequent and constant. Crystals are rare. Pulse of high tension, and heart is hypertrophied.

II. FROM DISEASES OF OTHER ORGANS.

1. LUMBAGO AND INTERCOSTAL NEURALGIA. In these: the pain is related to muscular effort. Generally bilateral. Muscles are fixed when the pain is present. Absence of destructive renal signs.
2. SPINAL CARIES.—In these: Fixidity, rigidity, tenderness, and pain of the spine. Urinary symptoms, if present, are associated with paraplegia.
3. BILIARY COLIC.—In this: Relation to jaundice. Signs of tenderness or enlargement of the gall-bladder.
4. APPENDICITIS of recurrent variety.—In this: Tenderness over McBurney's point. Tenderness per rectum. Intestinal symptoms.

5. GASTRIC OR DUODENAL ULCERS.—In these: Relation to food. Dyspepsia, vomiting.

In all the above, except sometimes in 2, there is an absence of any urine changes.

6. DISEASE IN THE BLADDER—growth or stone. In these: Vesical irritation. Pain after micturition. Blood comes at the end of micturition. Cystoscope shows the disease.

IN CASES OF UNCERTAINTY, with marked symptoms: Exploratory incision. Incision into capsule and parenchyma. Passage of ureteral bougie.

Prognosis.

DANGER FROM: Impaction in ureter. Cicatrization and stenosis of ureter. Atrophy of kidney. Hydronephrosis. Suppurative pyelonephritis. Perinephric inflammation, suppuration. Fistula. Affection of opposite kidney. Anuria.

AFTER OPERATION. — Nephrolithotomy: 2 per cent die. Nephrotomy: 23 per cent. Nephrectomy: 29 per cent.

Treatment of renal calculus.

PROPHYLACTIC.

EXERCISE and fresh air.

AVOID malt liquors, port, champagne, vinegar or salads containing it, rich meats, excess of butcher's meat, sweetbreads, liver, kidneys.

PLENTY OF DILUENTS: Distilled water, Contrexéville or Vichy waters.

DRUGS.

Alkalies, bicarbonates, potash, soda.
Citrate, tartrate, acetate of potash.
Carbonate and benzoate of lithia.
Piperazine, Sidonal, Urotropin } Diuretics, solvents of uric acid and antiseptics.

OXALURIA.—As above, but also:

AVOID hard water, green fruits, asparagus, tomatoes, rhubarb, gooseberries, grapes.

TONICS, e.g., Iron, strychnine, and quinine.

PHOSPHATURIA.—Treat neurasthenia and dyspepsia. Acids. Tonics.

SECONDARY CALCULI.—Aseptic precautions. Avoid mineral waters. Urotropin, etc.

PALLIATIVE TREATMENT.—For pain and colic: Morphia. Hot bath. Opium and belladonna fomentations. Alkaline drinks.

RADICAL OR SURGICAL TREATMENT.

NEPHROLITHOTOMY.—Operation of choice. Symptoms which justify it are: Hæmaturia, crystals and pus, constant pain or colic.

Renal Calculus—Treatment, *continued.*

NEPHROTOMY.—For calculous pyonephrosis, and all septic conditions complicating calculi.

NEPHRECTOMY.—Kidney completely destroyed. After nephrotomy has been repeatedly necessary. For intractable fistula following nephrotomy.

PARTIAL NEPHRECTOMY.—In suitable cases.

Lumbar incision when calculus is already in a fistula or perirenal abscess.

CALCULOUS ANURIA.

Etiology.

CAUSES : (1) Blocking of ureter by small stone ; (2) Destruction of kidney by large stone.

IMPLIES either only one functional kidney, or (rare) reflex suppression in the opposite kidney which has been injured.

POSITION OF IMPACTION.—Renal pelvis 13 per cent ; upper end of ureter 60 per cent; middle of ureter 13 per cent; lower end of ureter 20 per cent; bladder (large calculus) 4 per cent.

AGE AND SEX.—May occur at any age or either sex. Commonest in males over forty.

HISTORY.—Generally : antecedent history of colic. Sometimes : disease of one kidney (other than calculus). Rarely : Simultaneous blocking of both ureters, blocking ureter of single kidney.

IMMEDIATE CAUSE.—Some jolt, jar, or exercise.

Symptoms.

ONSET.—Pain (colic) on side last affected—subsides after one or two days. Or dull aching pain lasting throughout.

Rarely onset is insidious and without marked colic or pain.

Ineffectual dysuria, or polyuria. Anuria may be complete, incomplete, or intermittent.

TOLERANT STAGE.—Complete anuria may last seven or ten days, without uræmic symptoms.

If anuria ceases temporarily or permanently, a large quantity of low specific gravity urine is passed at once.

Anuria is rarely complete.

Urine possed in intervals is of low specific gravity, poor in urea, light colour. Rarely with blood, albumin, or casts.

Polyuria may occur and delay fatal result.

There may be constant desire to micturate.

Depression, insomnia, and digestive disturbances.

WHEN ASSOCIATED WITH HYDRONEPHROSIS.—Length of disease is much greater—Lasts fifteen to twenty-five days. Generally history of repeated attacks of colic and anuria. Tumour, etc., can be felt.

URÆMIC STAGE.—Pulse full, slow, irregular. Epistaxis and œdema (due to venous stasis). Temperature low.

Profuse perspiration, pruritus, etc. Profuse salivation and expectoration. Constant and profuse vomiting, with constipation and meteorism.

Contraction of pupils. Muscular tremors, rarely amounting to convulsion. Complete depression of body and mind. Incessant restlessness. Drowsiness or semi-coma. Respiration slow and irregular.

Prognosis.

In cases left to nature, 80 per cent die ; 20 per cent recover by passage of calculus.

In cases operated on, 50 per cent recover.

During tolerant stage, i.e., before contraction of pupils and muscular tremors, there is always a chance of recovery.

Prognosis is worse in relapsing cases.

Pathology.—On one side there is absence, atrophy, disorganization, or hydronephrosis of the kidney, or an old impacted calculus.

On other side : Enlarged, congested kidney. Rarely hydronephrotic. Impacted calculus in pelvis or ureter.

Diagnosis.

IN STRAIGHTFORWARD CASES.—Renal colic on one side. History of colic on the opposite. Anuria.

IN OTHER CASES.—One kidney may have been destroyed without pain. Patient may have forgotten which side the colic was at first. Palpation of ureter may show tenderness. Rectal or vaginal examination may feel the stone. Otherwise open loin on side last affected.

FROM OTHER CONDITIONS.

CANCER OF UTERUS, CANCER OF BLADDER, AND OTHER PELVIC TUMOURS (frequent causes of anuria) : Diagnosis by pelvic examination.

TRAUMATIC ANURIA : By relation to injury.

ANURIA FOLLOWING OPERATIONS on lower urinary tract : Relation to the operation. Rigors, malaise.

BRIGHT'S DISEASE.

Anuria is accompanied by headache, vomiting, coma, and convulsions. What urine there is, is dense, bloody, and solid with albumin. Secretion is resumed gradually.

AMYLOID DEGENERATION.—Long history of polyuria, œdema, etc.

HYSTERIA : other symptoms.

DRUGS (cantharides, turpentine, mercury) : History of their inception. Gradual onset. Urine bloody, and like Bright's disease.

RETENTION OF URINE is excluded by passing a catheter.

Calculous Anuria, *continued.*

Treatment.—Operate as soon as diagnosis is clear. Open kidney last affected—On tender side—On side of greatest abdominal resistance.

NATURE OF OPERATION.—To remove stone, if possible, by nephrolithotomy. To establish lumbar fistula if stone cannot be removed. In cases where stone can be located in lower ureter, then ureterotomy.

TUMOURS OF THE KIDNEY.

AGE.—Two periods of life they are commonest: (1) Under five years, and (2) Forty to fifty years.

VARIETIES.—Sarcoma, 40 per cent; carcinoma 25 per cent; cystic degeneration 15 der cent; others 20 per cent.

Distinctive Points of Kidney Swellings.

1. Colon in front of tumour: either resonant or felt as a roll.
2. No space or resonance between tumour and spine.
3. Do not bulge behind, but only cause a fullness of the loin.
4. Absence of any sharp edges.
5. Slight or no movement with respiration.
6. Generally do not cross mid-line; do not invade pelvis; have a line of resonance between them and the liver; except in extreme cases of hydronephrosis.
7. When they touch the anterior abdominal wall they do so about the level of the navel.
8. Varicocele of recent development is frequent.
9. Changes in urine or micturition.

Diagnosis from :—

LIVER TUMOUR OR ENLARGEMENT.—Moves with respiration. Fingers cannot get between it and ribs. No dullness in lumbar region. No intestine in front.

SPLEEN.—No bowel in front. Sharp edge—sometimes notched. Resonance between it and the spine. Moves with respiration.

SUPRARENAL.—Generally indistinguishable from renal. Generally not crossed by bowel.

OVARIAN.—Intestines behind. Grows from below upwards. Joined to the uterus. Loins are resonant. Urine is normal.

ENLARGED LYMPHATIC GLANDS.

FÆCAL ACCUMULATIONS.

MALIGNANT GROWTH OF LARGE INTESTINE.—Lacks shape of kidney. Symptoms referable to bowel. If large, is generally very fixed.

GROWTHS of mesentery, omentum, pancreas, gall-bladder.

PERINEPHRIC AND PARANEPHRIC TUMOURS: Bulge out into the loin. Mobility is very restricted. Cannot be felt anteriorly.

Malignant Disease of Kidney.

Etiology.—80 per cent cases are secondary; 20 per cent are primary. Large proportion of primary cases are due to injury or irritation of a calculus.

Pathology.

ADENOMA.—Spheroidal and encapsuled. Metastasis in lungs and bones. Proliferation of convoluted tubes. Spaces or tubes lined with renal epithelium. Adrenal "rest" tumours form most of these. Generally in adults, between forty and sixty.

CARCINOMA.—In old people fifty to sixty-five. Commoner on right side and in men. Two forms: capsulated and diffuse. Invades neighbouring viscera, especially colon or duodenum, and vena cava. Alveolar structure, with blood-cysts. Metastases in lungs and liver.

SARCOMA.—In children under five, and less often in adults, twenty to sixty. Grow to enormous size. Invade vena cava, ureter, and lymph-glands. Encapsuled in early stages. Hæmorrhagic and pseudo-cysts.

MYOSARCOMA occurs especially in infants. Probably congenital.

Symptoms.

HÆMATURIA.—Occurs in 80 per cent. First symptom in over 50 per cent. Spontaneous. Uninfluenced by repose or exertion. Profuse generally. Often occurs in intervals and lasts six days. Sometimes clot-moulds of ureter. Not so common in children.

TUMOUR.—First symptom in 25 per cent adult cases. First symptom in majority children. Often difficult to detect, especially in upper pole. Absence of tenderness usually. Jaundice, constipation, and vomiting sometimes occur when right kidney is affected.

PAIN.—Often absent in children. First symptom in 35 per cent adults. Begins in loin. Radiates to thorax, thighs, genitals. Uninfluenced by rest. Intermittent.

URINE.—Usually polyuria. Diminished urea and chlorides. Pus or albumin rare. May be frequent micturition.

VARICOCELE.—Comparatively sudden occurrence. Rapid development.

GENERAL SYMPTOMS.—Anæmia, emaciation, "cachexia." Appear late (one to two years) and develop rapidly.

	CARCINOMA.	SARCOMA.	
		ADULT.	INFANT.
Age ..	40–60.	20–40.	5.
Hæmaturia ..	Profuse in 70 %.	Seldom profuse.	Slight.
Pain ..	Great.	Great.	Slight.
Cachexia ..	Early.	Late.	Early.
Duration ..	3–3½ yrs.	5–6 yrs.	Less than 1 yr.
Metastasis ..	Usual.	Rare.	Rare.

Malignant Disease of Kidney, *continued.*

Diagnosis.

1. WHERE A TUMOUR IS PRESENT WITHOUT HÆMATURIA :—

   Malignant disease is probable : In children under ten and adults between forty and seventy. Rapid growth. No periodic lessening in size. Venous obstruction—Swelling of the leg or varicocele. Enlarged glands.

2. WHERE HÆMATURIA IS PRESENT, BUT NO TUMOUR :—

   (*a*) Renal Origin.—Blood thoroughly mixed with urine. Passage of worm-like clots. Cystoscopic examination.

   (*b*) Nature of Renal Disease.

   Calculus.—Movement provokes hæmorrhage. Colic.

   Tubercle.—Hæmaturia slight. Pyuria. Bacilli can be found. Patient is a young adult.

   Nephritis.—Casts. Albumin in excess of blood.

   Rare Conditions.—"Essential hæmaturia" or angeioneurotic cases. "Nephralgic hæmaturia." Hæmophilia. Movable kidney.

3. WHERE TUMOUR AND HÆMATURIA CO-EXIST :—

   Diagnosis from calculous disease and tuberculous disease (as above). Polycystic kidney—Polyuria, anuria, or uræmia ; generally double. Cysts. Hydronephrosis caused by tumour of bladder.

Treatment.

NEPHRECTOMY.—25 per cent of cases die, and of the 75 per cent recoveries the majority have recurrence within three years.

Transperitoneal for large growths. Lumbar for small.

A Combination of both incisions gives best results, because adhesions and pedicle can best be dealt with and drainage provided for.

Benign Tumours of Kidney.

ADENOMA.—Generally only size of pea and causes no symptoms. May have papillary and cystic formations.

FIBROMA.

LIPOMA, OSTEOMA, AND CHONDROMA.

ANGEIOMA, LYMPHANGEIOMA, AND MYOMA.

CYSTS OF THE KIDNEY.—(1) Retention cysts in granular kidney ; (2) Dermoid cysts (very rare) ; (3) Simple cysts ; (4) Conglomerate cysts ; (5) Hydatid cysts ; (6) Paranephric cysts.

Simple Cysts.—Generally single, size varies from that of a cherry to a melon. At one pole of kidney. Filled with watery or colloid material. No symptoms except pressure. Treatment by partial nephrectomy.

The Large Polycystic Kidney.

Pathology.—Weigh 1 to 16 lb. Retain kidney shape. Multitude of cysts from pin's head upwards. Lined by cubical epithelium. Contain fluid or colloid material. Almost always bilateral. Often associated with cystic disease of liver and spleen. Hypertrophy of left ventricle common. In infants it is associated with various congenital defects.

THEORIES OF ORIGIN.—Inclusion of mesonephric elements (Wolffian body). Failure of union between excretory tubes and ducts. Feebleness of tubules, which render them liable to dilatation. Cirrhosis or sclerosis, obstructing tubules. New growth, e.g., colloid adenoma.

Etiology.—Either in infants: "congenital cystic abscess," or between ages of twenty and eighty. Males more often than females.

Symptoms.

VERY INSIDIOUS ONSET, resembling chronic Bright's disease.

PAIN.—Dull aching in loins. Sense of weight and discomfort. Some tenderness.

TUMOUR on one or both sides in about 50 per cent.

GENERAL SYMPTOMS.—Anuria or polyuria sometimes. Intermittent albuminuria or hæmaturia.

Urine resembles that of chronic Bright's—Pale, low specific gravity, few casts, variable albumin. Nausea, vomiting, flatulence, etc. Headache, delirium, convulsions, coma.

Cardiac and pulmonary symptoms, dyspnœa, bronchitis, etc.

Cachexia in only few cases.

Diagnosis.—Very difficult. Combination of tumours with symptoms of chronic Bright's.

Prognosis.—Slow progress towards uræmia (one to ten years).

Treatment.—Nephrectomy if only one kidney be affected, and only if that is causing great pain or bleeding.

Tumours of Renal Pelvis.

COMMONEST VARIETIES.—(1) Simple papilloma; (2) Epithelioma. Simple may become malignant.

Piece of growth may become detached and grafted on ureter or bladder. Often cause hydro- or hæmatonephrosis. Vary from size of pea to huge mass. Often associated with and due to calculus.

DIAGNOSIS generally impossible from tumours of renal parenchyma.

Presence of villous tufts in urine when cystoscope shows bladder normal.

SYMPTOMS.

TUMOUR generally occurs, and is due to hydro- or hæmatonephrosis. Sometimes tumour alternates with hæmaturia.

Tumours of Renal Pelvis, *continued.*

Hæmaturia.—Earliest symptom. May be slight, intermittent, or fatal.

Urine.—May have albumin or pus. Varies in amount.

Pain.—Unusual. May be colic due to passage of clots. May be due to distention.

General.—Anæmia, etc. Uræmia (late).

TREATMENT.—Nephrectomy or nephro-ureterectomy.

SUBCUTANEOUS INJURIES OF URETERS.

Etiology.—Result generally from crushes, kicks, or falls. Possibly from compression of ureter against transverse vertebral processes. Possibly from a violent falling forward of the kidney. Often in young people (below twenty-five).

Pathology.—Ureter torn right across or lacerated. Peritoneum generally not torn.

If peritoneum is torn, urine is extravasated into peritoneal cavity.

If the tear in the ureter is extra-peritoneal, a urinary extravasation into loin occurs.

If the injury does not tear through, but obstructs ureter, some form of nephrectasis results.

Symptoms.

HÆMATURIA.—Slight, severe, or absent.

URINE.—May be suppressed if both kidneys are injured.

PAIN.—As in kidney injuries.

TUMOUR.—If patient survives the accident, either an abdominal or loin tumour results.

If tumour is due to extravasation outside kidney, it appears in a few days.

If tumour is due to nephrectasis, it only develops in weeks.

Suppuration occurs in tumour generally.

Diagnosis.—Is only apparent after the formation of the tumour following an injury.

Prognosis.—Is only bad when associated with severe injury to both kidneys, or rupture of the peritoneum.

Treatment.—Puncture is unsatisfactory.

Lumbar Incision and drainage is the best routine treatment.

Suture Ureter if rent can be found. Do not ligature proximal end of ureter.

Nephrectomy should only be a last resort in case of severe suppuration or intractable fistula.

Operation Wounds of Ureter.

Commonest in pelvic portion of the ureter. Near cervix uteri, or in the midst of adhesions or malignant growth.

Results either in cutaneous or vaginal fistula, or peritonitis, or localized peritoneal urinary abscess.

Treatment.

URETERO-URETERAL ANASTOMOSIS.

DANGER is that of stenosis resulting from cicatrization.

METHODS: (1) End to end—very careful and minute stitching; (2) End in end—unsatisfactory; (3) Lateral implantation—best; (4) Oblique end to end.

PRECAUTIONS.—Ends should be lightly ligatured. Search made for stone or stricture. Catheter retained in bladder. Drain retroperitoneal cases.

URETERAL GRAFTING.—When ureter is out of reach for minute plastic operations. When some length of ureter has been removed. Best into bladder. If not long enough for this, into colon rather than rectum or small intestine.

VAGINAL GRAFTING.—When time is of great importance, and when proximal end of ureter is long enough.

CUTANEOUS GRAFTING.—Through a stab-wound in the loin.

In all grafting methods there is great danger of septic infection of the kidney owing to the absence of the natural ureteral sphincter.

LIGATION OF BOTH ENDS OF THE URETER.—When time will allow no other method.

NEPHRECTOMY should only be secondary for relieving fistula or hydro- or pyonephrosis.

URETERITIS.

Primary.—Very rare. Calculus or injury.

Secondary.—Ascending—Descending—From pelvic inflammation. From mouth of a ureteral fistula.

Ascending Ureteritis.—Two factors: Distention—Inflammation.

CAUSES.—Those of cystitis and retention of urine. Also pelvic inflammation, uretero-vaginal fistula, pregnancy.

ANATOMY.—Three varieties:—

ACUTE.—Septic inflammation of mucous membrane. Little or no dilatation or induration.

CHRONIC DILATED FORM.—Ureter may be size of small intestine. Great tortuosity at both ends. Valves form at the bends. Canal is tortuous and obstructed. Little or no peri-ureteritis. Great increase of muscular tissue of ureter. Often cysts in mucous membrane.

FIBROUS TYPE.—Intimately adherent dense mass of peri-ureteral tissue. Ureter straight, thickened, and indurated. All coats inflamed, with excess of fibrous tissue. Strictures are long, rigid, and almost impermeable, or may be annular. Often associated with single pyonephrosis.

CONDITION OF URETERO-VESICAL ORIFICE.—Generally contracted. Sometimes dilated. Valve is destroyed by: (1) Mechanical effects of retention; (2) Inflammatory effects of cystitis.

OBSTRUCTION OF URETER BY VALVES OR STRICTURE.

Causes of Valve Formation.—Temporary obstruction (e.g., pregnancy, etc.) causes kinking and dilatation of renal pelvis. Dilated pouch of renal pelvis may press on the ureter. Inflammatory dilatation in uretero-pyelitis. Enfolding of mucous membrane round a stone.

Causes of Strictures.

CONGENITAL.—Generally close to renal pelvis. By aberrant blood-vessels.

ACQUIRED.—Injury. Cicatrices following ulceration. Impaction of calculus. Ureteritis.

Symptoms.—Commonest in females about thirty and in right ureter.

TUMOUR, either kidney or ureter.

PAIN recurrent at intervals of weeks. Acute resembling renal colic. Dull aching in loin in intervals.

URINE may contain PUS, or CRYSTALS, or BLOOD.

TENDERNESS on bimanual examination, reduction of tumour, and filling of bladder.

BLADDER.—Frequency of micturition. Inability to retain water. Pain on passing water.

STRICTURE can sometimes be felt per vaginam, or felt with the ureteral catheter.

Treatment.—Exploratory nephrotomy. Pass bougie from above, if possible. Pass bougie from below.

1. If stricture or valve exist at junction of pelvis and ureter: Longitudinal incision. Sewn together as transverse incision.
2. If the valve is caused by ureter opening high up into dilated pelvis: Divide ureter and sac down to lowest point, and sew edges together.
3. When a high oblique junction of ureter to hydronephrosis coexists with a stricture: Excise the stricture. Unite the lower end of ureter with lowest part of dilated pelvis. Split the end of the ureter before sewing it in its new position (Küster's operation).
4. When stricture is in the pelvis: Ureterotomy with plastic operation, or nephrectomy.
5. Lumbar fistula.
6. Colpo-ureterotresis—grafting the ureter into the vagina.
7. Uretero-ureteral anastomosis, or grafting into the opposite ureter.
8. Nephro-ureterectomy or nephrectomy, where ureter is past mending.

CALCULUS IN URETER.

Etiology.—In great majority of cases renal in origin. May form round foreign body, e.g., blood-clot. May remain in situ indefinitely, or be discharged within eighteen months, and kidney resume its functions.

SITUATION.—(1) Just below commencement of ureter; (2) At vesical orifice (especially in women); (3) At brim of pelvis.

Symptoms.—Colic. Pain shooting along ureter. Tenderness over ureter. Hæmaturia. Anuria (if opposite kidney is blocked or diseased). Abdominal tumour (persistent or intermittent). Urine contains crystals, blood, or pus. Prolapse of ureter into bladder. Skiagram shows stone.

Diagnosis.—In the majority of cases the symptoms are simply those of renal calculus, and diagnosis is only complete on lumbar exploration and passing ureteral sound.

In some cases the stone can be felt per rectum or vaginam.

FROM ENCYSTED VESICAL CALCULUS: impossible clinically.

FROM CYSTITIS.—In this: Urine is alkaline. Pus at beginning or end of micturition.

FROM URETERITIS.—Difficult in absence of history of colic.

FROM VESICAL TUBERCULOSIS.—In this: Tubercle in urine. Polyuria. Frequent micturition. Slight hæmaturia. Symptoms not relieved by rest.

FROM PROLAPSED INFLAMED OVARY.—Ovary lies behind broad ligament and at greater distance from vaginal wall. Stone is felt in antero-lateral fornix. Hardness and outline are more definite.

THE SKIAGRAM shows a shadow in the course of the ureter, but this has to be distinguished from that of calcareous iliac glands or that of an appendix concretion (the latter very rarely throws a shadow).

URETERAL CATHETERIZATION through the cystoscope not only shows which side is affected, but the end of the catheter may touch the stone and a waxed tip receives a scratched impression.

Prognosis.

KIDNEY.—Atrophy if block is complete and permanent. Recovery if block is removed within six to eighteen months. Hydronephrosis if block is incomplete. Pyonephrosis if sepsis occurs.

LIFE.—Not affected if other kidney is good. Endangered by Inflammation and suppuration of kidney, extravasation of urine, anuria if opposite kidney is bad.

Pathological Effects.

ON URETER.

ABOVE.—Thinned, dilated, sacculated, and tortuous. Or œdematous, inflamed, and ulcerated with rupture.

AT THE POINT OF IMPACTION.—Ulceration and stricture.

BELOW.—Unchanged, or thickened and stenosed. Prolapse of ureteral orifice. Ulceration of stone into bladder.

Calculus in Ureter—Pathological Effects, *continued.*

ON PERI-URETERAL TISSUE.—Induration, inflammation. Abscess or fistula.

ON KIDNEY.—Any variety of atrophy—Inflammation—Cystic kidney—Hydro- or pyonephrosis. Perinephritis and suppuration. Fistula.

Treatment.—Except when causing anuria (*vide supra*) the case should be kept under observation in order to note whether the stone is fixed or moving. In the latter case time should be given for natural expulsion.

IMPACTED NEAR THE KIDNEY.—Attempt to push back and remove through the renal pelvis. Failing this, removal through an incision in the ureter, which is then sewn up.

IMPACTED AT THE BRIM OF THE PELVIS.—Removal through a long oblique lumbar incision which is produced down in front of the iliac spine.

IMPACTED BELOW THE PELVIC BRIM.—Removal through a parasacral incision similar to that of Kraske's excision of the rectum.

IMPACTED IN THE BLADDER WALL.—Removal through the bladder by a suprapubic operation. In women it may be done through the vagina, but this involves the risk of a vaginal fistula.

RENAL INSUFFICIENCY.*

The incompetence of the kidneys to carry out their excretory functions. It may be absolute or only relative. Normally there exists more than twice as much kidney substance as is necessary for the vital economy. If by atrophy, disease, or blocking of the ureters some of the kidney substance is destroyed or rendered functionless, a limit is reached beyond which any further kidney loss will be fatal. This is a matter of great importance in all operations upon the kidneys, ureters, prostate, or bladder, because either by direct removal of kidney substance or by nerve shock the line between relative and absolute insufficiency may be overstepped. The following are the methods available for the estimation of the sufficiency of both kidneys or of one.

THE AMOUNT OF THE URINE AND ITS CONSTITUENTS.

QUANTITY varies from 500 to 700 ccm.

UREA varies from 12 to 15 grams.

CHLORIDES vary from 5 to 6 grams.

Any diminution of these quantities below 30 % indicates the lowest limit of renal sufficiency.

THE AMOUNT OF UREA FROM EACH KIDNEY when the urine is collected separately by ureteral catheterization is 1·2 gram in 2 hours. Anything below ·75 gram indicates serious insufficiency.

METHYLENE BLUE TEST.—This pigment may be given as a pill, or better as an intramuscular injection (15 min. of a 5 %

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* Thomson Walker, *Lancet*, Vol. I, 1907, p. 711.

solution). Normally it appears as a colourless chromogen in the urine fifteen minutes after the injection, the urine becoming blue on boiling with acetic acid. The blue itself appears about half-an-hour later. Its elimination ought to cease in twelve to twenty-four hours. Renal insufficiency is indicated by: (1) A late appearance of the chromogen and the blue in the urine, and (2) Prolonged elimination lasting more than three days.

CHROMOCYSTOSCOPY. — Indigo - carmine is injected intramuscularly (4 cc. of a 4 % solution), and in normal cases should be visible as a red stream through both ureteric orifices within twenty minutes, being best seen thirty minutes later. The absence of one kidney or a blocked ureter are obviously demonstrated by this method.

THE PHLORIDZIN TEST.—Subcutaneous injection is given of 15 min. of a 0·5 % solution of phloridzin. Normally sugar can be recovered from the urine about thirty minutes later, and its excretion continues for four hours, the amount recoverable varying from 0·5 to 2·5 grams. Its excretion is diminished or delayed in renal insufficiency, but as this also occurs in comparatively trivial renal disease its value is less than that of other methods.

CRYOSCOPY.—The freezing-points of the urine and of the blood are constant within certain limits. They are altered in renal inadequacy. These freezing points are known as the cryoscopic index, and are indicated by the sign $\triangle$.

THE NORMAL $\triangle$ FOR URINE is −1·3° C. to −2·3° C.
A raised index indicates inadequacy.

THE NORMAL $\triangle$ FOR BLOOD is −0·56° C.
A lowered index indicates inadequacy.

CHAPTER XLIII.

THE DIAGNOSIS OF ABDOMINAL DISEASE.

ACUTE ABDOMINAL AFFECTIONS.

IT is of the utmost importance that a clear idea should be obtained of the *general nature* of acute intra-abdominal lesions ; it is often impossible to obtain any precise knowledge of the locality, position, and nature of the lesion until the abdomen is opened. In many cases, to wait for the signs necessary for a complete diagnosis will postpone treatment until it is useless. It is, for example, the utmost folly to wait for the full clinical picture of a ruptured gastric ulcer to be developed. A reasonable suspicion of such an event makes instant laparotomy imperative. There are other cases in which opening the abdomen subjects the patient to an unnecessary operation, but otherwise does but little harm, e.g. in renal or lead colic. And again there exists a small group of cases in which an exploratory operation may turn the balance of the scale in a fatal direction. Such are cholera or acid intoxication. Thus the nature of the lesion with reference to the necessity for laparotomy should be the first and chief object sought, and for this purpose the following classification will be useful.

Classification of acute abdominal lesions.

I. SUDDEN PERITONEAL INVASION.—The peritoneal cavity is more or less suddenly attacked by the outpouring of septic or irritating fluid.

SIGNS AND SYMPTOMS common to all this group are :—

1. Sudden and very severe pain felt all over the abdomen.
2. Profound shock, which has little or no tendency to recovery, but slowly merges into the collapse of peritonitis.
3. Intense tenderness and rigidity of the abdominal wall.

VARIETIES : Ruptured stomach, duodenum, small intestine, large intestine, appendix, gall-bladder, urinary bladder, pyosalpinx, hydatid cyst, abscess ; acute "idiopathic" peritonitis, notably the pneumococcus variety.

II. HÆMORRHAGE INTO THE PERITONEAL CAVITY.—Here the loss of blood is the cardinal fact, whilst shock and peritoneal invasion are of minor importance.

SIGNS AND SYMPTOMS :—

1. Pain and shock are moderate and usually are strictly proportionate to the degree of traumatism, which is the common cause.

2. The signs of hæmorrhage :—pallor, restlessness, rapid compressible pulse, and sighing respiration.
3. A steady progress of symptoms, e.g., rising pulse-rate and increasing distention, is the most noteworthy sign.
4. Evidence of free fluid in the flanks or pelvis.

VARIETIES : Ruptured liver, spleen, kidney, uterus, gravid tube, blood-vessels.

III. INTESTINAL OBSTRUCTION.—Here the peritoneal cavity is not affected at first, and shock therefore passes off before peritonitis sets in.

SIGNS AND SYMPTOMS :—

1. Constipation is usually absolute from the first.
2. The vomiting of shock, which is often the first symptom in all acute abdominal lesions, merges into that incessant vomiting characteristic of obstruction.
3. Tenderness and abdominal rigidity are absent in the early stages.

VARIETIES.—Hernia, internal strangulation, volvulus, mesenteric thrombosis, obstruction by foreign bodies.

IV. TORSION, STRANGULATION, OR ACUTE INFLAMMATION OF A VISCUS.—Here the signs will be varied and indefinite, but more definitely localized than in the other groups.

SIGNS AND SYMPTOMS.

1. Shock is of less degree and more gradual onset than in the other groups.
2. Pain is severe, but usually preceded by premonitory symptoms of an intermittent nature.
3. Definite localization is possible, because the seat of the pain is over some abnormal swelling or area of resistance.

VARIETIES.—Acute intussusception, twisted renal pedicle, twisted splenic pedicle, twisted ovarian tumour, acute pancreatitis, torsion of the testis, strangulation of a solid viscus in a hernia, e.g., the ovary or omentum.

V. COLIC.—Here there is no primary inflammatory lesion, but merely pain due to distention of a duct or abnormal muscular spasm.

SIGNS AND SYMPTOMS.

1. Agonizing pain predominates the case. This is usually localized over the affected part and often has a characteristic radiating distribution, and is intermittent. It begins suddenly and ends suddenly.
2. Although the abdomen is often rigid, pressure on it relieves rather than aggravates the pain.
3. There is usually a history of some antecedent condition which is the cause of the colic, e.g., jaundice, urinary symptoms, or contact with lead.

VARIETIES.—Biliary colic, renal colic. [illegible] s or other gut colic.

Acute Abdominal Affections—Classification, *continued.*

VI. NON-SURGICAL CONDITIONS, which may simulate any of the above. These hardly permit of any general description, except that the vomiting, diarrhœa, or pain are not correlated to signs of abdominal disease in the way which occurs in the other groups.

VARIETIES.—Cholera, acute irritant poisoning, crises of tabes dorsalis, basal pleuro-pneumonia, acute acid intoxication.

History.—The existence of any preceding illness or pain, and the exact character of this, are of the utmost importance.

DYSPEPSIA.—Pain directly after food in gastric ulcer. Pain one or two hours after food in duodenal ulcer. Indefinite digestive disturbances may precede pancreatis, biliary colic, or almost any acute lesion. Occasionally a gastric and much more frequently a duodenal ulcer may perforate which has been quite latent.

CONSTIPATION of marked degree and increasing severity precedes rupture of a stercoral or malignant ulcer.

DIARRHŒA precedes the rupture of a tuberculous or dysenteric ulcer.

JAUNDICE in cases of gall-stone colic, ruptured gall-bladder, intestinal obstruction by gall-stones, and some cases of pancreatic disease.

DISORDERS OF MICTURITION.—Difficult, frequent, or painful micturition often precedes renal colic, renal crises, or ruptured urinary bladder.

PREVIOUS ATTACKS OF PAIN generally occur in the various forms of colic, biliary, renal, or lead, also in displaced kidney or spleen, or twisted ovarian cyst. Appendicitis also may have existed as recurrent attacks, but the fulminating attack which gives difficulty in diagnosis is usually the first.

LEAD HABITS, e.g., painting or colour-making, precede lead colic.

TRAUMATISM, if severe and direct, suggests a ruptured viscus. If less severe and indirect it may be the cause of a twisted viscus, biliary or renal colic, or a ruptured extra-uterine gestation.

MENSTRUAL HISTORY gives indications as to the possibility of some form of abnormal pregnancy or rupture of a pregnant uterus.

General Symptoms.

PULSE is of increased rate in most cases. A normal pulse practically excludes all cases of ruptured viscera, peritonitis, intestinal obstruction, twisted viscera, or hæmorrhage, and points to the case being one of colic. A small, hard wiry pulse is suggestive of sudden peritoneal invasion, and a rapid and compressible pulse of hæmorrhage. The slowing of the pulse indicates the passing off of shock; its increase in rate signifies a progressive lesion, and is of gravest import when associated with a falling temperature.

TEMPERATURE is of very little value in early diagnosis, because it is so often practically normal in the gravest conditions, e.g., ruptured ulcer, intestinal obstruction, or colic. A high temperature which remains high suggests peritonitis of the less virulent type. An intermittent high temperature with rigors indicates suppuration in the biliary ducts or possibly portal pyæmia. A low temperature indicates collapse or sudden peritoneal invasion or hæmorrhage. A sudden drop from a high to a sub-normal temperature accompanies the perforation of a typhoid ulcer.

VOMITING as an initial symptom is of no significance, as it accompanies any condition of shock. If it persists, continues, and increases, it indicates obstruction. Coming on late it suggests peritonitis. Blood-stained vomit is seen in stomach injuries and rarely in perforated ulcer. But continued vomiting is against the diagnosis of a gastric lesion, and the worst cases of perforated gastric ulcer never vomit at all.

CONSTIPATION of some degree accompanies most acute abdominal lesions. If after the lower bowel has emptied itself it is absolute and lasting, it indicates obstruction or peritonitis.

DIARRHŒA with blood and mucus accompanies intussusception, colitis, cholera, enteric fever, and irritant poisoning.

PAIN is common to all these cases. Its characters are important.

VERY SUDDEN ONSET: Ruptured viscus, biliary or renal colic.

ACUTE PAIN PRECEDED BY PREMONITORY SYMPTOMS: Strangulated hernia, intestinal obstruction, twisted viscus, pancreatitis.

DIFFUSE, but chiefly umbilical, is the common type in peritoneal invasion and obstruction.

LOCALIZED: Above the navel in gastric ulcer, pancreatitis, ruptured liver, gall-stone diseases, or ruptured spleen. Right iliac fossa in appendicitis. Hypogastrium in tubal gestation, pyosalpinx, or ruptured bladder. In the loin and referred to the inguinal region in twisted kidney and renal colic. Over the right rectus in gall-stones and duodenal ulcer.

OF INTERMITTENT CHARACTER in various forms of colic, in partial obstruction, and in acute intussusception.

The Abdomen.

MOVEMENT is seldom quite natural.

FREE MOVEMENTS in colic, intestinal obstruction (early).

IMPAIRED MOVEMENTS in twisted viscus or pelvic lesions.

NO MOVEMENT characterizes all forms of peritoneal invasion, ruptured viscus, and hæmorrhage of traumatic origin.

RIGIDITY goes with impairment of movement, and gives more detailed information, thus :—

RIGIDITY OF THE UPPER ABDOMEN follows lesions of stomach, liver, gall-bladder or duct, pancreas, and spleen.

RIGIDITY OF THE LOWER ABDOMEN goes with appendicitis and pelvic lesions.

RIGIDITY MORE MARKED ON THE RIGHT SIDE with biliary, appendix, and duodenal lesions.

Acute Abdominal Affections—The Abdomen, *continued.*

TENDERNESS and rigidity generally go together. A marked absence of tenderness, the patient pressing on his abdomen for relief, is suggestive of renal, biliary, or lead colic, or intestinal obstruction. The most acute tenderness is seen in cases of peritoneal invasion and ruptured viscus.

DISTENTION is rare as an early sign. Coming on within a few hours of the onset it indicates a ruptured viscus, effusion of gas, fluid, or blood, and is then associated with tenderness and rigidity, or acute intestinal obstruction ; especially volvulus, when rigidity and tenderness will be less marked.

PERCUSSION.

SHIFTING DULLNESS in the flanks, especially if it increases in a few hours, indicates free fluid, blood, or visceral contents, poured out from a ruptured viscus.

OBSCURED LIVER DULLNESS of rapid development indicates free gas from a ruptured stomach or gut. But if present from the outset it may be due to a distended colon. A normal liver dullness excludes free peritoneal gas.

TUMOUR is rarely present, and more rarely palpable. It is most significant in acute intussusception, being sausage-shaped, over the colon, and hardening during the attacks of pain. Displaced kidney or spleen, ovarian tumours, ruptured uterus, or distended gall-bladder may be felt.

Pelvic Examination.

INSIDE THE RECTUM.—An intussusception or new growth may be felt. Marked ballooning suggests sigmoid disease or some form of pelvic peritonitis.

OUTSIDE THE RECTUM.—Right-sided inflammatory swelling is felt in many appendicitis cases. Or a hæmatocele may indicate a ruptured tube.

BY THE VAGINA.—An ovarian or tubal swelling, and the condition of the uterus, may be determined.

Progress of the Case.—This is of the utmost importance in the acute cases.

RAPID INCREASE OF SYMPTOMS, especially the pulse-rate, rigidity, and distention, indicate a visceral lesion. This is seen within one or two hours, and demands immediate laparotomy.

STEADY INCREASE, especially of distention and vomiting, indicates intestinal obstruction.

STEADY IMPROVEMENT is seen in shock without visceral lesion after injuries.

IMPROVEMENT FOLLOWED BY RISING PULSE-RATE indicates hæmorrhage coming on after the subsidence of shock.

SUDDEN RELIEF OF PAIN occurs after the passage of a calculus, and, in a less degree and later in the case, in the occurrence of gangrene in strangulation of intestine or other viscus.

Special Examination in Anomalous Cases.

THE BASE OF THE CHEST may show evidence of pleurisy or pneumonia, which causes pain very like that of abdominal disease.

THE URINE.

Crystals, blood, casts, and albumin, suggest renal colic.
Bile pigments suggest biliary colic.
Sugar—very rarely in pancreatitis.
Indican—acute obstruction of the small intestine.
Cammidge's crystals—in pancreatitis.

THE URINE contains acetone or di-acetic acid in cases of ACID INTOXICATION. This occurs generally in fat children, vomiting being the chief symptom. A sweet smell in the breath should give the warning of the existence of this condition. Its recognition is of great importance, because an exploratory operation under an anæsthetic will probably end fatally.

THE FÆCES contain blood and mucus in cholera, colitis, and intussusception. Large quantities of fat and undigested meat fibres in pancreatitis. Blood intimately mixed with the fæces in duodenal ulcer.

DIAGNOSIS OF CHRONIC ABDOMINAL DISEASE.

This group, including as it does a great number and variety of different conditions, presents the greatest difficulty in diagnosis ; but in no district of the body is diagnosis more important, because the majority of the conditions are amenable to operative treatment. The diagnosis must always be accurate enough to answer the three questions: whether, when, and where to open the abdomen ? It is worse than futile to do a laparotomy for obesity or spinal caries. Delay may be fatal in operating for carcinoma of the stomach or gut. And the operation will do more harm than good if, e.g., a suppurating kidney is opened through the peritoneal cavity. But in many cases it is impossible to do more than suspect the probable nature of a disease, so that it is of great importance to examine every case with the utmost thoroughness, ascertaining every possible fact before operation, but being prepared to find the nature of the disease quite different from that suspected, during the operation. To put the matter in another way, it is necessary to avoid two extremes—on the one hand, that of not troubling to diagnose the condition, but subjecting every abdominal condition to operation ; and on the other, refusing to open the abdomen in cases when certainty as to the nature of the case is impossible without exploration.

Classification.

1. GENERAL ABDOMINAL DISTENTION.—Uniform enlargement. Fat, fluid, flatus, fæces. Phantom tumour. Large growth filling the abdomen.

Diagnosis of Chronic Abdominal Disease—Classification, *continued.*

2. LOCAL SWELLING.

(*a*) PARIETAL.—Epi-, inter-, or sub-parietal, according to its position outside, in, or inside the abdominal muscles.

(*b*) VISCERAL.
Hepatic
Biliary
Splenic
Gastric
Enteric
Colic
Omental
Pancreatic
Renal
Peri-renal
Uterine
Ovarian
Broad ligament
Testicular
Vesical.

(*c*) HERNIAL.—A viscus protruding through the parietes.
Inguinal
Femoral
Umbilical
Ventral.

3. LOCAL DISEASE WITHOUT SWELLING.

DISORDERS OF DIGESTION with disease of :—
Œsophagus
Stomach
Duodenum
Small gut
Large gut
Appendix
Liver
Bile-ducts
Pancreas.

JAUNDICE WITH ALTERATIONS IN THE FÆCES in diseases of :—
Liver
Gall-bladder
Bile-ducts
Pancreas
Duodenum.

DISORDERS OF MICTURITION WITH ALTERED URINE in diseases of :
Kidneys
Ureters
Bladder
Liver and bile-ducts
Pancreas
Abscesses connected with the urinary organs.

ALTERED BLOOD in :—
Some splenic tumours
Pyogenic abscesses
Tuberculous lesions (opsonins)
Cancer of stomach.

PAIN AS THE SOLE SYMPTOM :—
Colic : Biliary calculus—Renal calculus—Lead colic—Appendicitis—Ovaritis—Dysmenorrhœa.
Displaced viscera : Enteroptosis — Kidney — Spleen — Ovary.
Diseases of spine and nerves : Aneurysm—Caries—Tabes.

Conditions of General Abdominal Distention.

FAT, FLATUS, AND FÆCES give unaltered percussion, and bimanual examination detects no mass in the loins or pelvis.

FLUID, i.e. ASCITES, gives shifting dullness in the flanks, with resonance in the uppermost parts of the abdomen.

LARGE TUMOUR FILLING THE ABDOMEN gives dullness in the central parts, with resonant areas around it.

PHANTOM TUMOUR is in the lower part of the abdomen. No altered percussion. No tumour felt bimanually. Disappears under an anæsthetic.

Examination of a Local Swelling.

THE RELATION OF THE SWELLING TO THE PARIETAL WALL.—Movement of the swelling in relation to movements of the parietes, together with the effect of the contraction of the muscles upon the swelling, prove its position upon, in, under, or unconnected with the parietes.

EPIPARIETAL TUMOURS are quite unaffected by muscular contractions, e.g., lipomata or sebaceous cysts.

INTERPARIETAL SWELLINGS are hardened and fixed by muscular contractions, e.g., a fibroma or abscess in the muscles.

SUBPARIETAL SWELLINGS are obscured by muscular contractions, but if fixed to the parietes they always move with them, e.g., an inflammatory or malignant growth attached to the abdominal wall.

VISCERAL SWELLINGS, if unconnected with the parietes, allow the latter to move over them, and parietal muscular contraction tends to obliterate their outline.

HERNIAL SWELLINGS are situated at the linea alba, navel, femoral or inguinal regions, and become more prominent on muscular exertion.

THE POSITION OF THE SWELLING usually corresponds with the normal position of the affected viscus, thus :—

IN THE EPIGASTRIUM. — Gastric, or more rarely hepatic, swellings.

RIGHT HYPOCHONDRIUM.—Liver, gall-bladder, right kidney and adrenal.

LEFT HYPOCHONDRIUM.—Spleen, left kidney and adrenal.

UMBILICAL.—Pancreatic, omental, and small intestine.

LUMBAR.—Renal and pararenal. Colon.

RIGHT ILIAC REGION.—Appendix, cæcum. Psoas, iliac, or pelvic abscesses.

LEFT ILIAC REGION.—Sigmoid colon. Psoas, iliac, or pelvic abscesses.

HYPOGASTRIUM.—Uterus, bladder. Ovarian tumours.

OCCUPYING A LARGE PART OF THE ABDOMEN.—Ovarian, uterine, omental tumours. Hydronephrosis. Malignant and tuberculous disease.

MOVEMENT OF VISCERAL SWELLINGS IN RELATION TO THAT OF THE PARIETES.

SWELLINGS WHICH MOVE WITH THE PARIETES deep in the abdomen are probably inflammatory or malignant.

MOVEMENT WITH RESPIRATION is most marked in hepatic, splenic, and gastric swellings, less so with renal.

MOBILITY INSIDE THE ABDOMEN.

HEPATIC AND GASTRIC GROWTHS have little mobility apart from respiratory movements.

A WANDERING SPLEEN can be moved about, with the left hypochondrium as a centre.

GALL-BLADDER SWELLINGS permit sometimes of lateral movements round a centre at the hepatic margin.

Diagnosis of Chronic Abdominal Disease—Local Swelling, *continued.*

- Renal Swellings may be moved up and down, but not at all laterally.
- Ovarian and Uterine Swellings can be moved from side to side, but not upwards away from the pelvis.
- Fixed to the Posterior Parietes are pancreatic cysts, aneurysm, and deep malignant disease.

OUTLINE OF THE SWELLING.

- A Sharp Edge characterizes hepatic and most splenic swellings.
- A Notched Edge is only found in the spleen.
- A Craggy Nodular Mass is found in malignant growths.
- Intense Hardness suggests actinomycosis or cancer.
- A very Ill-defined Edge suggests inflammatory or malignant invasion of the peritoneum.
- A Globular Outline suggests a cyst—renal, ovarian, pancreatic, etc.

PERIODIC CHANGES IN SIZE AND CONSISTENCY.

- Peristaltic Contractions occur in the stomach or gut above an obstruction.
- Painful Contractions with Hardening occur in an intussusception, also in a pregnant uterus at labour.
- Irregular Movements, seen or felt, occur in the borborygmi of obstruction and as fœtal movements.
- Distended Bladder disappears on catheterization.

PERCUSSION gives valuable information as to: (1) The relation of the alimentary canal to the swelling; and (2) the presence of free fluid or gas in the abdomen or swelling.

- Swellings which are Resonant are gas-containing abscesses, e.g., subphrenic and rarely parametric abscesses. Also dilated parts of the stomach or gut, and posterior (pancreatic) tumours covered by stomach and bowel.
- Swellings which are Dull form the majority of visceral tumours and displacements.
- Kidney Swellings have an area of colic resonance in front of them.
- Hepatic, Biliary, and Splenic Swellings have the colic resonance below them.
- Ovarian Cysts are generally dull and surrounded by a resonant area above and at the sides.
- Ascitic Collections are resonant centrally, with dull flanks.

Symptom Groups in Abdominal Disease.

DIGESTIVE.

- Anorexia, Vomiting, and Dyspepsia, in disease of the stomach, duodenum, and pancreas.
- Constipation occurs in any form of obstruction, whether in the stomach or gut. Also in inflammatory conditions, e.g appendicitis, and in pelvic tumours or displacement pressing on the rectum.

Diarrhœa Alternating with Constipation characterizes chronic obstruction in the large bowel.

Passage of Blood and Mucus is found in malignant disease of the large bowel, and in intussusception, dysentery, and colitis.

JAUNDICE, WITH COLIC AND PALE STOOLS, occurs in hepatic, bile-duct, and pancreatic disease.

DISORDERS OF MICTURITION in renal disease, stone, or displacement; vesical, prostatic, and urethral diseases. Also in disease, displacements, and tumours in the pelvis.

ALTERATIONS IN THE URINE.—Blood, pus, crystals, casts, parasitic ova, mucus, in urinary disease. Bile in hepatic, biliary, and pancreatic disease. Cammidge's crystals in disease of the pancreas.

MENSTRUAL IRREGULARITIES.

Amenorrhœa in normal and abnormal pregnancy.

Menorrhagia in uterine disease.

Irregular Menstruation in extra-uterine pregnancy.

Dysmenorrhœa in pelvic inflammatory disease and disease of the uterine appendages.

LOCOMOTIVE DISORDERS.

Increase of Pain caused by walking, in renal and vesical calculus. Diseases of the spine.

Ataxia, with loss of knee-jerks, etc., in tabes.

VASCULAR DISORDERS.

Arteriosclerosis, high-tension pulse, cardiac hypertrophy, accompany an Aneurysm.

STEADY LOSS OF WEIGHT is of extreme significance, and generally results from progressive, i.e., malignant, disease of the alimentary canal. The higher the disease the greater and more rapid is this sign. Chronic gastric ulcer and chronic pancreatitis also cause marked emaciation.

SYMPTOMLESS SWELLINGS.—Cysts of the ovary or omentum. Hydatid cysts. Uterine fibroids. Tuberculous abscesses, arising from bones (e.g. the ilium), without involving joints. Gummata of the abdominal wall, liver, and spleen.

Special Methods of Examination in Obscure Cases.

OF THE STOMACH.—Distention with air and fluid, illumination, and shadow of a bismuth meal.

RADIOGRAPHY shows disease of the spine or pelvic bones, aneurysm. After bismuth meals it may show the form and position of the stomach and large intestine. Calculi in the kidney, ureter, or bladder. Certain foreign bodies.

DISTENTION OF THE COLON WITH AIR shows whether a growth is in or outside the colon, and emphasizes the relations of the renal, hepatic, biliary, and splenic tumours.

CYSTOSCOPY, SEGREGATION OF URINE, AND U[illegible] CATHETERIZATION, demonstrate obscure les[illegible] kidneys, ureters, or bladder, especially tuberculosis.

CHAPTER XLIV.

AFFECTIONS OF THE BLADDER.

ECTOPIA VESICÆ.

Anatomy.—Bladder is only represented by the trigone bearing the ureteral orifices and part of the posterior wall. This part of the bladder is placed in the anterior abdominal wall in the hypogastrium. The penis is rudimentary and shows complete epispadias. The pelvic bones are so ill-developed that the pubes do not meet at the symphysis. The pelvis is very shallow from before backwards. The umbilicus is not recognizable as such.

Symptoms.—Constant dribbling of urine. Excoriation of the skin. Eventual pyelonephritis.

Treatment.

1. Implantation of the vesical trigone into the rectum, upon which it lies. This does not open the peritoneum.
2. Division of the sacro-iliac joints—forcing the pubes together. Subsequent plastic operation to cover bladder.
3. Plastic operations by which skin flaps are sewn over the bladder cutaneous surface inwards.
4. The provision of a vulcanite cup to form a urinal.

RUPTURE OF THE BLADDER.

Causes.—Direct blows on the distended bladder. By a wound from a portion of fractured pelvis. Penetrating wounds. Bursting following retention. Bladder is generally ulcerated or sacculated.

Varieties.—(1) Intraperitoneal; (2) Extraperitoneal.

Intraperitoneal Rupture.—Superior surface is involved. Severe shock.

Painful, scanty, blood-stained micturition.

SIGNS OF FREE FLUID in the abdomen.

PERITONITIS.

SIGNS.—Catheter withdraws only scanty bloody urine. If lotion is injected into the bladder, less returns than is inserted.

TREATMENT.—Laparotomy. Sponge out the free fluid. Sew up the bladder wound. Drain rectovesical pouch if the urine is septic. Tie in a large catheter.

Extraperitoneal Rupture.—Involves the base or lateral walls. Micturition may not at first be interfered with.

PELVIC CELLULITIS or EXTRAVASATION OF URINE occurs.

DUSKY SWELLING occurs above the pubes and in the perineum.

RAPIDLY FATAL TOXÆMIA if the urine is septic.

TREATMENT.—Free incisions into the swollen areas above pubes and in the perineum. Drain the bladder through a catheter, or by a median urethrotomy.

CYSTITIS.

Acute Catarrh.

CAUSES.—Cold. Uric acid diathesis. Gout. Irritants, e.g., cantharides.

Acute Septic Cystitis.

CAUSES.—Traumatism—operation, foreign body. Gonorrhœa. Septic wounds or catheters. Direct infection with the colon bacillus through the female urethra. All the causes of chronic cystitis are predisposing causes of acute cystitis.

Chronic Cystitis.

CAUSES.—Stone. Tumour. Large prostate. Stricture. Paralysis from spinal disease. Tuberculosis.

Bacteriology.

Bacillus coli (alone)	In acid urine.
Tubercle ,,	
Gonococcus ,,	
Streptococcus ,,	
Bacillus proteus	Alkaline urine.
Diplococcus ureæ	
Staphylococci	
Bacillus coli with the above	

Symptoms.

PAIN in hypogastrium and perineum.

VESICAL IRRITABILITY.—Constant desire to micturate. Frequent micturition. Pain after micturition.

URINE.

In Acute Cases.—May contain blood—Shreds and sloughs of the mucous membrane.

In Some Cases.—If infection is purely by an acid-forming organism (*B. coli*, *gonococcus*, tubercle), not much change is apparent. The organism can be recognized.

In Majority of Cases.—Urine becomes alkaline by a formation of ammonia from the decomposition of urea. Foul smell. Loaded with pus, mucus, epithelium, and phosphates.

GENERAL SYMPTOMS (chiefly in the acute cases).—Toxæmia. Low febrile condition. Exhaustion from want of sleep.

COMPLICATIONS.—Septic infection of the kidneys.

Anatomy.

ACUTE CHANGES.

Catarrh. Acute inflammation of the mucous membrane.

Mucous Membrane is intensely congested. Its vessels become thrombosed. Patches slough and leave ulcers. It may be cast off as a complete slough.

Cystitis—Anatomy, *continued.*

CHRONIC CHANGES.

MUCOUS MEMBRANE, especially over the trigone.—Thickened and covered with tenacious mucopus mixed with phosphatic débris. Superficial layers lost. Congestion, with large dilated vessels. Sacculated and ulcerated.

MUSCULAR WALL.—At first thickened and hypertrophied. Later (if obstruction exists), becomes dilated, thinned, and fasciculated.

URETERS.—Orifices are contracted by muscular spasm.

KIDNEYS.—Hydronephrosis. Pyonephrosis. Pyelonephritis.

Treatment.

GENERAL.—In acute cases: Rest in bed. Milk diet.
Remove the cause (e.g., stone, stricture).

DRUGS.—Copious alkaline drinks (to diminish the irritating uric acids). Belladonna and hyoscyamus—for spasm.

ANTISEPTICS.—Urotropin, salol, benzoic acid, to prevent ammoniacal decomposition and to retard the growth of organisms.

DIURETICS.—Citrate of potash, buchu, to diminish the concentration of the urine.

OLEO-RESINS.—Sandalwood oil, cubebs, copaiba, etc. In chronic cases. When there is much mucopus, and especially in chronic gonorrhœa.

LOCAL APPLICATIONS (to be avoided in acute cases).

Irrigation with dilute solutions of permanganate of potash and boroglyceride.

In very chronic cases: protargol gr. ij ad ℥j.

CYSTOTOMY.—In intractable cases, and as a preliminary to further operations. Perineal route provides the best drainage. Suprapubic route is most convenient if a tumour is going to be removed.

TUMOURS OF THE BLADDER.

Varieties.

BENIGN.—Papilloma—Fibroma and myxoma (rare).

MALIGNANT.—Epithelioma—Columnar-celled carcinoma (secondary to uterine or rectal disease)—Sarcoma (rare).

Papilloma or Benign Villous Tumour.—Occurs in patients from fourteen to forty. Men more often than women.

Anatomy.—Tuft of branching processes. Each consists of central connective tissue and blood-vessels, clothed with transitional squamous epithelium. Submucous and muscular tissues are not involved. Generally at or near the trigone and close to one of the ureteric orifices. Occasionally multiple. May be secondary to villous tumour of the renal pelvis.

Symptoms.—Three stages: (1) Latent, (2) Hæmaturia, (3) Cystitis.

HÆMATURIA.—So-called symptomless hæmaturia, from the absence of other signs or symptoms. Profuse hæmorrhage, more marked at end of micturition. Recurs at long intervals, and lasts for a few days at a time.

VESICAL IRRITABILITY.—Frequent micturition. Pain after micturition from the growth being caught in the orifice of the urethra.

CYSTITIS.—Chronic variety. Passage of quantity of ropy mucus.

HYDRONEPHROSIS (occasional), from a blocking of the ureteric orifice.

Signs.—Sound produces sharp hæmorrhage. Nothing felt by rectum or vagina. Cystoscope shows growth.

Treatment.

PALLIATIVE.—Sulphuric acid dil. by mouth, injections of suprarenalin, protargol, or silver nitrate solutions into the bladder.

OPERATIVE.—Suprapubic cystotomy. Isolate growth with a Fergusson's speculum. Press up its base from the rectum or vagina. Cut away the mucous and submucous tissue from which it grows.

Prognosis.—There is often recurrence with ultimate malignant development.

Epithelioma.

OCCURS in patients over forty. Men more often than women.

Begins in two ways:—

1. AS A BENIGN PAPILLOMA, which subsequently becomes malignant, by growing down into the submucous and muscular tissue.
2. AS A TYPICAL MALIGNANT ULCER infiltrating the deep tissues from the first.

SITUATION.—Generally just above the trigone, but may invade the whole bladder wall.

HÆMATURIA.—Most marked in the case of a villous growth.

CYSTITIS.—Vesical irritability, accompanied by great pain in hypogastrium and perineum.

COMPLICATIONS.—Hydronephrosis with pain in the kidney from involvement of the ureter. Pyelonephritis and septicæmia. Retention of urine (rare), from involvement of the urethra.

Signs.—Indurated mass felt in the bladder wall, per rectum or per vaginam. Cystoscope shows a sessile papilloma or an ulcer with thickened margins. Sound may feel a rough area on the bladder wall.

Tumour of the Bladder, *continued.*

Treatment.

1. SUPRAPUBIC CYSTOTOMY.—Exposure as in benign growth. Removal of a wide area of bladder wall, including its whole thickness.

   If the ureter is involved, the affected part is removed and the proximal end implanted into the bladder in a fresh situation.

2. TOTAL EXTIRPATION OF THE BLADDER.—In extensive growths. Ureters implanted in the rectum or colon. Or a double nephrostomy performed.

Diagnosis of Vesical Growths.

1. FROM OTHER CONDITIONS RESEMBLING IT.
   - (*a*) RENAL DISEASE.—Stone or new growth. In this: Pain or tumour in the loin. Hæmorrhage is much less profuse. Blood is evenly mixed with the urine. Segregation shows difference between the two sides. Urine remains acid. There is less vesical irritability.
   - (*b*) STONE IN THE BLADDER.—In this: Pain after micturition, with spasm, is present. Bleeding is slight. Sound feels the stone.
   - (*c*) TUBERCLE OF THE BLADDER. In this: Pain and vesical irritability are severe. Bleeding is absent or trivial. Tubercle bacilli in the urine. Cystoscope shows shallow undermined ulcers.
2. OF THE VARIETY OF GROWTH.

   BENIGN GROWTHS are characterized by: Youth of the patient. Profuse recurrent hæmaturia without other symptoms at first. Absence of induration of the bladder wall.

   MALIGNANT GROWTHS are characterized by: Patient is over forty-five. Pain and cystitis are early and prominent. Induration is felt in the vesical wall.

STONE IN THE BLADDER.

Varieties.

URIC ACID.—Oval or spherical stone. Brown in colour and laminated in texture. Deposits as rhomboidal, "wheatsheaf," or irregular clumps of crystals.

In acid urine, as result of excessive sweating, too little drinking, excess of nitrogenous food, sedentary habits, febrile condition, gouty diathesis. Forms much the commonest variety of stone.

AMMONIUM URATE.—Similar to the above, but lighter in colour. Deposits as spiculated globules or as an amorphous deposit.

CALCIUM OXALATE.—Rough, nodulated like a mulberry. Very hard. Dark brown or blackish.

Deposits in acid urine, as "envelope," or dumb-bell-shaped crystals. In nervous and dyspeptic patients.

PHOSPHATIC CALCULUS.—Usually a secondary deposit on a uric acid or oxalate stone or foreign body. Sometimes primary in a saccule of a bladder deposited by cystitis. Forms a white, soft, friable mass.

Deposits in alkaline urine as triple phosphate (ammonio-magnesium), "knife-rest" crystals, or hexagonal feathery crystals. Calcium phosphate. Amorphous deposit. The mixed phosphates form a "fusible calculus."

CYSTIN.—Yellow, waxy—very rare.

XANTHIN.—Red—very rare.

Structure.

NUCLEUS at the centre formed by foreign body, renal calculus, or mucus.

BODY.—Concentric layers of crystals held together by mucus.

CRUST.—Most recent deposit. Generally the mixed phosphates. Occurs when cystitis with alkaline urine has been set up.

NUMBER.—Usually single. Rarely multiple.

Etiology.

AGE.—Any age, but especially children under ten; men past middle life.

SEX.—Men much more often than women.

SOCIAL CONDITION.—In children it chiefly affects the poor.

CLIMATE, SOIL, etc.—Very common in India and the tropics. Common in Eastern Counties and where the water is very hard.

LOCAL URINARY CONDITIONS.—Any obstruction predisposes to the deposit of a calculus: especially phimosis in children, and a large prostate in old men.

Gout, lithiasis, or oxaluria usually precede and cause it, often first causing a renal calculus.

Symptoms.

PREMONITORY.—Renal colic. Incontinence of urine (in children). Passage of gravel, uric acid, or oxalates.

PAIN.—In bladder and perineum. Referred to the end of the penis. Worse at the end of micturition.

VESICAL IRRITABILITY.—Constant micturition, followed by pain and spasm, with sensation of not having emptied the bladder.

HÆMATURIA.—Slight and at the end of micturition.

INFLUENCE OF MOVEMENT.—All symptoms aggravated by movement, and therefore worse by day than night.

SUDDEN CESSATION OF MICTURITION, or

ACTUAL RETENTION of urine, from impaction of the stone at orifice or in the urethra.

EFFECTS OF STRAINING and nerve irritation.—Piles or prolapse. Hernia. Priapism.

IN CHILDREN.—Incontinence of urine. Constant pulling at the foreskin.

Stone in the Bladder, *continued.*

Signs.

SOUNDING.—Bladder should be full of urine or lotion. Buttocks raised. Anæsthetic should be given in children. Beak is turned from side to side and then backwards behind a large prostate.

Hard click indicates a stone. It can be heard and felt. Most marked in oxalate or uric stones. Least marked in phosphatic stones. May be masked by mucus.

FALLACIES in sounding for a stone:—

Stone may be missed because: It lies deep behind a large prostate, or in a saccule with a narrow mouth, or it is covered with tenacious mucus.

Click or grating is given by conditions other than a stone, viz.: Prostatic calculus. Phosphatic débris on an ulcer or new growth.

PALPATION.—Large stone can be felt by rectum or vagina bimanually.

Diagnosis.—From renal calculus, vesical new growth, vesical tuberculosis (*vide supra*).

INCONTINENCE IN CHILDREN.—Stone should always be suggested by marked priapism or intractability of the symptoms.

Course and Complications.—In old men it may long remain latent. In children it quickly causes symptoms. Cystitis occurs before long.

KIDNEY CHANGES.—Hydronephrosis—from vesical spasm. Pyonephrosis. Pyelonephritis from ascending infection.

Treatment.

PRELIMINARY FACTS to be ascertained.

1. Presence of any urinary obstruction. Size and position of stricture. Presence and character of prostatic growth.
2. Size of the stone. By palpation through the rectum. By grasping with a lithotrite.
3. Nature and consistency of the stone. By examination of the crystals in the urine. By the smooth, or rough, or soft sensation imparted to a sound.
4. Presence of cystitis or kidney disease.

LITHOLAPAXY.

INDICATIONS.—Stone less than 1½ in. in diameter. Stone must be friable. Absence of stricture or very large prostate. Absence of cystitis or kidney complications.

METHOD.—Buttocks raised. Bladder filled with boracic lotion (at least 6 oz.). Grasp the stone and crush it by lithotrite. Crush all fragments that can be grasped. Wash out all fragments by the evacuator. Use the largest evacuating tube possible. Again sound for and treat any residual fragments.

SUPRAPUBIC CYSTOTOMY.

INDICATIONS.—Large stone (1½ in. or more). Hard stone which cannot be crushed (especially oxalate stones). Combined with a large prostate. When a stricture exists. When the stone is encysted.

METHOD.—Trendelenburg position. Fill the bladder until it is felt above the pubes. Open immediately above the pubes. After removal of the stone: Drain if cystitis or hæmorrhage exists; sew up the bladder, but drain the wound otherwise.

LATERAL LITHOTOMY.

INDICATIONS.—A contracted bladder. Severe cystitis. When a stricture (traumatic) of the membranous urethra exists and can be dealt with at the same time.

METHOD.—Pass a sound with a lateral groove into the urethra. Fill the bladder. Place the patient in the lithotomy position. Assistant holds sound in mid-line and against the pubes. Cut from 1½ in. in front of anus to a point midway between anus and left tuber ischii. Deepen this until the knife is in the groove of the sound. Push knife forward into the bladder along the groove. Insert finger and forceps and extract the stone. Leave in a large tube to drain the bladder.

INCONTINENCE OF URINE.

Definition.—Involuntary micturition due to abnormal detrusor action or defective sphincter action.

1. **Active Incontinence.**—Abnormal detrusor action, common in children, especially boys. Generally at night—"nocturnal incontinence."

CAUSES.—Reflex stimulation of abnormally excitable vesical centre. For example, by: Slight vesical distention—Rectal parasites or polypus—Phimosis—Uric acid gravel—Emotions.

TREATMENT.—Remove any abnormal cause of irritation.

Wake the child to pass water before the bladder is too full.

Bland diet: No meat in evenings.

Drugs: Iron and arsenic as tonics. Belladonna and bromides as sedatives.

Instruments (in obstinate cases only): Passage of a metal sound. Gentle faradization through this.

2. **Passive Incontinence.**—Defective sphincter action.

(*a*) PARALYTIC.—From injury of vesical centre in the cord. Sphincter is paralyzed and remains relaxed. Urine dribbles out from the ureters to the urethra. Bladder remains empty and contracted.

(*b*) MECHANICAL.—When a stone or growth holds open the neck of the bladder (very rare). Over-distention of female urethra.

TREATMENT of passive incontinence is that of its cause. Also an attempt to prevent cystitis by aseptic precautions at the external meatus.

Incontinence of Urine, *continued.*

3. **False Incontinence.**—Distention with overflow. Bladder is greatly distended, and the urine constantly dribbles away.
    (*a*) OBSTRUCTION to the outflow of urine. Enlarged prostate. Stricture of the urethra.
    (*b*) SPINAL CORD INJURY.

TREATMENT is that of the cause, with aseptic precautions.

RETENTION OF URINE.

Causes.

1. OBSTRUCTION TO THE OUTFLOW.
    - IN PENIS.—Phimosis—Rings, bands round penis—Malignant growth (very rarely causes obstruction).
    - IN URETHRA.—Stricture—Congestion with spasm—Impacted stone—Rupture.
    - IN PROSTATE.—Hypertrophy—New growth—Inflammation.
    - IN BLADDER.—New growth—Stone (rarely cause retention).
    - OUTSIDE NECK OF THE BLADDER.—Pressure of tumours, e.g., uterine fibroids—Retroverted gravid uterus.
2. PARALYSIS.—Diseases of the spinal cord, especially tabes or myelitis. Injury of brain or spinal cord. Injury of the reflex nerve mechanism, e.g., by operations on the rectum or genital organs. Hysteria.
3. ATONY of the bladder produced by: Over-distention—acute or chronic. Cystitis (rare). In old age (probably associated with prostatic enlargement).

Symptoms.—Bladder presents as a tense hypogastric swelling. Pain of a constant character in abdomen and perineum (this is absent in paralytic cases.)

Results.

CLINICALLY.—(1) Rupture of the bladder; or (2) Rupture of the urethra; or (3) Distention with overflow—false incontinence.

PATHOLOGICALLY.

- ON THE BLADDER.—In acute cases: Dilatation and atony. In chronic cases: Hypertrophy, followed by dilatation and sacculation.
- ON THE KIDNEYS.—Hydronephrosis or pyonephrosis. Pyelonephritis.

Diagnosis.

SUPPRESSION OF URINE.—In this: The bladder is empty and contracted. Follows some severe shock, or operation, or nephritis. Scanty urine is of high specific gravity and albuminous.

CALCULOUS ANURIA.—History of lumbar pain and colic. Bladder is empty. Pain is absent or exists only in the loins.

RUPTURED BLADDER.—Follows an injury. Bladder is empty or contains bloody urine. More lotion can be put in than got out of the bladder.

Treatment.

1. WHEN CONGESTIVE SPASM IS THE CAUSE: First try hot baths, with morphia and belladonna suppositories. Then pass a soft catheter.
2. WHEN DEFINITE OBSTRUCTION EXISTS: Pass large soft catheter first; if this fails, then small or filiform catheters.
3. WHEN THE PROSTATE IS ENLARGED: First try large soft catheter, then catheter coudé, then metal prostatic catheter.
4. WHEN NO KIND OF CATHETER CAN BE PASSED:—

SUPRAPUBIC ASPIRATION.—Pass a soft catheter through the cannula and leave it.

SUPRAPUBIC CYSTOTOMY.—When a large prostate or vesical growth can be dealt with later, or calculus at the same time.

PERINEAL CYSTOTOMY.—When a urethral stricture exists which can be treated at the same time.

IN ACUTE RETENTION OF MANY HOURS' STANDING, and
IN CHRONIC RETENTION WITH CYSTITIS, it is better to relieve the retention first by a catheter or aspiration, and do a radical operation later when the patient and the bladder are in better condition.

CHAPTER XLV.

DISEASES OF THE PROSTATE.

INFLAMMATION.

Varieties.—Acute prostatitis, acute abscess. Chronic prostatitis. Tuberculous prostatitis.

Acute Prostatitis.

CAUSES.—Gonorrhœa. Sepsis secondary to instrumentation—stricture—Cystitis.

ANATOMY.—Inflammatory swelling of the whole organ. Ducts and follicles plugged by mucus and bacteria. Abscess forms: (*a*) In one of the follicles; or (*b*) In the parenchyma spreading through the capsule.

SYMPTOMS.—Burning pain in perineum, worse on sitting. Frequent painful micturition. Retention of urine from spasm and congestion. Constitutional symptoms of fever. Constipation. Pain during and after defæcation.

SIGNS.—Passage of prostatic shreds or pus in urine. Inflamed prostate felt per rectum. Brawny swelling in the perineum. When suppuration occurs under or outside the capsule, fluctuation felt by rectum or in the perineum.

COMPLICATIONS.—Abscess may burst into: Urethra—Bladder—Rectum—Perineum. Rectal or perineal fistula may result. Cystitis and septic diseases of the kidneys.

TREATMENT.—Rest and milk diet. Hot hip baths. Fomentations to perineum. Hot enemata. Morphia and belladonna suppositories. For retention: Soft catheter. For suppuration: Median perineal incision.

Chronic Prostatitis.

CAUSES.—Posterior urethritis (gonorrhœa). Cystitis. Stricture.

SYMPTOMS.—Frequent micturition. Slight pain after micturition. Sense of weight in the perineum. Discharge of albuminous material after micturition, especially the first thing in the morning. Passage of mucous casts of the prostatic glands. Rarely a chronic abscess forms.

SIGNS.—Tender enlargement felt per rectum.

COMPLICATION.—Formation of prostatic calculi.

TREATMENT.—Bland diet. Absence of sexual excitement. Copious fluids, with diuretics. Irrigation of the prostatic urethra with protargol lotion, gr. j ad ℥j. Passage of large bougies. Belladonna suppositories.

Tuberculous Prostatitis.—Usually secondary to tuberculous disease of testis or vesiculæ seminales. Rarely primary.

ANATOMY.—Caseous deposits in the prostate. Chronic abscesses. Ragged ulceration, generally the result of septic infection.

COMPLICATIONS.—In all except the mild primary cases it spreads to the bladder and kidneys.

SYMPTOMS.—Those of chronic prostatitis. Pyuria, with urine which is acid at first.

TREATMENT.—Generally only constitutional. Bland diet with copious fluids. Morphia and belladonna suppositories for pain. Rarely in primary cases, removal through the perineum.

Prostatic Calculi result from deposit of lime salts in the follicles. In cases of chronic prostatitis. Consist of carbonate of lime. Usually multiple. Collect in enlarged prostatic pouches.

SYMPTOMS.—Irritability of the bladder. Difficulty in micturition.

SIGNS.—Those of chronic prostatitis. Felt by a sound just before entering the bladder.

TREATMENT.—Removal through a median perineal incision.

ENLARGEMENT OF THE PROSTATE.

Anatomy of the Normal Gland.—Shaped like a pyramid, with four triangular surfaces. Normally measures a little over an inch transversely, about one inch in other diameters.

SURFACES.

SUPERIOR SURFACE is in the cavity of the bladder; is pierced by the internal meatus; is covered by vesical mucous membrane.

POSTERIOR SURFACE is related to vesiculæ seminales. Separated from rectum by fascia and cellular tissue, and is covered by rectovesical fascia.

LATERAL SURFACES.—Lie on the origin of the levatores ani from the pubes. Are covered by visceral layer of pelvic fascia. This is connected to the pubis in front as the puboprostatic ligament.

APEX.—Situated on the junction between the lateral surfaces. Is pierced by the urethra. Rests on that part of the pelvic fascia which forms the deep layer of the triangular ligament.

CONNECTIONS.

BLADDER.—The mucous membrane is continued over its superior surface into the urethra. The muscular wall is continued into its posterior and lateral surfaces.

URETHRA traverses the gland from its superior surface to its apex.

EJACULATORY DUCTS.—Traverse its substance from its posterior surface to join the urethra.

Enlargement of Prostate—Anatomy of Normal Gland, *continued.*

SURROUNDINGS.—The gland with its fibromuscular capsule is surrounded by:

1. The neck of the bladder, which is structurally continuous with the fibromuscular capsule.
2. Cellular tissue containing the prostatic plexus of veins.
3. A sheath of fascia. (1) Deep layer of triangular ligament. (2) Visceral layer of pelvic fascia at the sides. (3) Recto-vesical fascia behind. (These are all parts of the same fascia, forming a continuous "SHEATH" for the prostate).

STRUCTURE.—Consists of two kinds of tissue: (1) Mass of unstriped muscle and fibrous tissue; (2) Glandular tissue embedded in (1).

CAPSULE consists of the peripheral parts of the fibromuscular tissue arranged in a laminated manner. This dips down as a median raphé, and joins the anterior and posterior walls of the urethra. There is no distinct demarcation between the capsule and the rest of the gland in normal conditions.

LOBES.—Two primary lateral lobes lie on each side of the median raphé. A central, median, or third lobe is that part of the gland superior and posterior to the internal urethral meatus, and is divided from the lateral lobes by the ejaculatory ducts.

DUCTS.—The glands of the lateral lobes open by ten to fifteen ducts into the groove on the floor of the urethra at the side of the crista urethræ. The glands of the middle lobe open into a recess—the sinus pocularis—which opens on the summit of the crista urethræ. The ejaculatory ducts open on either side of the orifice of the sinus pocularis. The median lobe is the uterus masculinus.

Etiology.—Common after fifty. May be caused by prostatitis, urethritis, or lithiasis, or any condition of chronic vesical irritability. Usually is merely a senile change.

Anatomy.

PROSTATIC CHANGES.

SHAPE AND SIZE.—At first it retains its shape, then it becomes lobulated and irregular. The lateral lobes enlarge towards the rectum.

The median lobe and the whole of the superior surface grow up into the bladder.

A median constriction marks the attachment of the neck of the bladder, and separates the intravesical from the extravesical parts of the gland.

STRUCTURE.—The enlarged gland consists of either: (1) Excess of fibrous tissue: usual in the smallest specimens which cause symptoms; (2) Overgrowth of all the normal tissues: usual in the smooth, medium-sized specimens; (3) Fibromyomatous tumours or fibroids: may form large masses; (4) Fibro-adenomata, i.e., overgrowth of the glandular tissue:

constitute the bulk of the largest specimens; (5) A well-marked laminated capsule consisting of layers of fibro-muscular tissue surrounds the rest of the mass, and is continuous with the muscular walls of the bladder.

CONSISTENCY.—Hard when chiefly fibrous tissue. Soft when formed by adenomata.

CHANGES IN THE URETHRA.—Elongated from one to several inches. Curved with the convexity backwards. Tortuous from the pressure of enlarged lobules. Internal meatus may be distorted or blocked by the growth of the median lobe, or rarely held open by the same.

CHANGES IN THE BLADDER AND KIDNEYS.—The bladder becomes first hypertrophied and then dilated, the muscular fasciculi being separated, and the mucous membrane pouched between the fasciculi. Cystitis occurs sooner or later. Hydronephrosis, pyonephrosis, or pyelonephritis may result from chronic retention and cystitis.

Symptoms.—There are usually four stages, though the earlier ones may not be noticed or complained of, viz. :—

1. DIFFICULTY IN MICTURITION.—A long time is taken in starting the stream. The stream is of good bulk but slow, and projected with little or no force. Straining often checks the stream.
2. FREQUENT MICTURITION.—Due at first to congestion and irritability of the neck of the bladder, later to retention. Worse at night, or more noticeable then.
3. RETENTION OF URINE.—The bladder cannot empty itself when the obstruction increases or when the bladder becomes weak. Hence some residual urine is left after each act. This varies from one to ten ounces, and constitutes the measure of the inefficiency of the bladder. Acute retention may be the first sign, or more commonly supervenes on chronic retention when micturition has been delayed so long that the bladder becomes atonic, or results from congestive spasm caused by cold or alcoholic excess. False incontinence (retention with overflow) may supervene in any neglected case.
4. CHRONIC CYSTITIS.—Infection is usually by instruments. May arise by auto-infection. Urine becomes ammoniacal and loaded with phosphates, pus, and mucus.

Physical Signs.

SIGNS OF RETENTION OF URINE in the absence of stricture or paralysis. Bladder felt above the pubes (rare in early cases). Presence of residual urine after micturition.

RECTAL EXAMINATION.—Gland is felt on the anterior rectal wall, about 2½ in. from the anus. It feels large or lobulated. It may feel practically normal.

Enlargement of Prostate—Physical Signs, *continued.*

URETHRAL EXAMINATION.—The prostatic urethra is elongated —judged from the distance the catheter must be passed from the pubic arch till the bladder is entered. Normally this is about 1½ in., in prostatic cases it is anything up to 4 in.

A long curved catheter or a coudé pass most easily.

CYSTOSCOPE.—The intravesical enlargement may be seen with difficulty.

Treatment.

1. DIET AND DRUGS.—For mild cases without any residual urine.

DIET.—Meat and rich food to be taken very sparingly. Plenty of milk, alkaline waters, and diluents. Avoid alcohol.

REGULATION OF THE BLADDER.—Never postpone micturition after the desire occurs.

DRUGS.—Alkalies, e.g., bicarbonate and citrate of potash. Sedatives, e.g., hyoscyamus or belladonna. Antiseptics: urotropin, salol, to prevent or minimize cystitis.

2. CATHETER TREATMENT.—For cases in which retention occurs, either acutely or chronically, shown by the existence of residual urine.

CHOICE OF INSTRUMENT.—(*a*) No. 12 rubber catheter; (*b*) Coudé or bicoudé; (*c*) Metal catheter with long prostatic curve. A rubber or metal instrument is better for regular use because it can be boiled.

METHOD.—Passed once a day—preferably at night. Antiseptic precautions for the meatus and catheter. Efficient lubrication.

CONTRA-INDICATIONS.—Existence of a tight urethral stricture. Great pain on catheterization, due to nervousness or irritability of the bladder. Bleeding on catheterization. Patients who cannot carry out the necessary antiseptic precautions. Patients who cannot pass the catheter themselves. Occurrence of rigors and fever (catheter fever). Cystitis (if this can be cured by irrigation of the bladder, the catheter may be resumed, but cystitis quickly recurs).

3. RADICAL OPERATIVE TREATMENT.—PROSTATECTOMY.

INDICATIONS.—Cases where catheter life is contra-indicated (*supra*). Comparatively young patients (fifty to sixty-five). When the prostate is rapidly growing (adenomata). When cystitis has occurred. (It is of course far better to operate before this).

SUPRAPUBIC OPERATION—the routine method.—Fill the bladder with lotion. Open above the pubes. Incise the mucous membrane over the prostate. Press up the prostate by left fingers in the rectum. Insert the index finger between the bladder wall and the prostate through the opening in the mucous membrane. Sweep round the prostate between the layers of the capsule. Tear through the urethra as it enters the deep layer of the triangular ligament. Remove the prostate. Irrigate the bladder with hot water. Tie in a large drainage tube. Sew the bladder and parietes round the tube. Arrange some suction or siphonage apparatus, e.g., Cathcart's irrigator or Colt's dressing. Patient should pass water by penis in seven to ten days. Lessen the drainage gradually.

DISADVANTAGES OF THE OPERATION.—The whole of the prostatic urethra is removed. The ejaculatory ducts are necessarily, and the vesiculæ seminales occasionally, removed.

RESULTS.—(*a*) Complete recovery, with normal micturition, in about 70 to 80 per cent. (*b*) Death in about 10 per cent of the cases from: Shock and hæmorrhage—Mania —Septic infection, especially through the kidneys— Senile asthenia. (*c*) Recovery, with permanent suprapubic fistula. (*d*) Recovery, with an atonic bladder which requires occasional catheterization.

PERINEAL PROSTATECTOMY.

INDICATIONS.—Slight enlargement. Severe cystitis. Contracted bladder. Feeble constitution.

METHOD.—Crescentic incision in front of the rectum. Deepened through the triangular ligament and the compressor urethræ. Cut through the deep layer of the triangular ligament, i.e., the visceral layer of pelvic fascia. Cut the urethra through as high as possible. Shell out the prostate through an incision into its capsule. Drain through the incision.

ADVANTAGES.—Less shock than the suprapubic method. The urethra can be more deliberately and accurately divided. Better drainage is afforded.

DISADVANTAGES.—Injury to the compressor urethræ and to the pelvic fascia. Greater liability to a permanent fistula.

4. OTHER OPERATIVE PROCEDURES.

Removal of adenomata from within the fibromuscular capsule of the prostate.

Removal of projecting masses through the urethra.

Castration or double vasectomy to induce prostatic atrophy.

These last two methods are only of historical interest.

MALIGNANT DISEASE OF THE PROSTATE.

This is always a round-celled carcinoma. Its ordinary symptoms and signs are indistinguishable from those of benign prostatic enlargement, and in its early stages it is seldom recognized as malignant before operation. The following points may indicate its nature.

1. The rapidity of development of the symptoms of urinary obstruction.
2. The early occurrence of hæmorrhage, both spontaneous and after instrumentation.

Malignant Disease of the Prostate, ***continued.***

3. Pain and cystitis will be earlier and more conspicuous than in simple enlargement.
4. By the rectum the growth feels large, hard, and nodular, and later the rectal walls become fixed to it.

TREATMENT.—If its nature is not recognized before suprapubic enucleation is performed, the operation may prove of exceptional difficulty, the growth tearing instead of shelling out. Bleeding will be unusually profuse, and recurrence early and rapid.

PERINEAL PROSTATECTOMY should be performed if malignancy is recognized before operation. After exposure of the gland and division of the membranous urethra, the prostate is removed, together with its proper capsule, from inside its sheath of pelvic fascia. The bladder walls are cut at their junction with the prostate, and the latter is removed. The bladder is sewn to the urethra over a rubber catheter.

CHAPTER XLVI.

AFFECTIONS OF THE URETHRA.

TRAUMATIC RUPTURE.

Causes.—Falling astride. Blows on the perineum. Fracture of the pelvis.

Situation.—Membranous urethra, usually as it passes through the (superficial) triangular ligament.

Extent.—Partial, through the floor; or entire, through the whole circumference.

Symptoms.—Pain. Retention of urine.

Signs.—Ecchymosis and swelling of perineum, scrotum, and penis. Extravasation of urine if micturition is attempted. Bleeding from the urethra.

Result.—Fibrous stricture of a dense resilient character, or death from extravasation.

Treatment.

IF NO PERINEAL BLEEDING OR SWELLING OCCUR: Adopt no local treatment. Rest in bed. Passage of soft catheter.

IF PERINEAL SWELLING OCCURS: Open the urethra at once. Find the ends of the torn portion. Sew together over a soft catheter. Leave catheter in for one week.

IF EXTRAVASATION OF URINE HAS OCCURRED: Open the urethra in the perineum; pass a tube into the bladder. Make multiple incisions over the swollen parts. Immediate union of the ends of the urethra over the catheter in recent cases.

STRICTURE OF THE URETHRA.

Anatomy of the Normal Urethra.

1. PROSTATIC URETHRA.

LENGTH.—One inch or more.

DIAMETER.—Large, admitting No. 20 sound. About ⅓ in.

SHAPE.—Triradiate in section.

STRUCTURE.—Median ridge—the crista urethræ runs along the floor. On this opens mesially the sinus pocularis, and at the sides the ejaculatory ducts.

Lateral grooves into which the ducts of the lateral lobes of the prostate open.

DIRECTION.—Vertical.

STRICTURE.—Never occurs.

Stricture of the Urethra—Anatomy of the Normal Urethra, ***continued.***

2. MEMBRANOUS URETHRA.

LENGTH.—Half an inch.

POSITION.—Runs from the prostatic sheath, i.e., the deep triangular ligament, to the superficial triangular ligament.

SURROUNDED by the compressor urethræ. Cowper's glands lie on each side.

DIAMETER.—About ¼ in. Admits a No. 12 sound with some tension. Narrows in its passage through the superficial triangular ligament.

DIRECTION.—Downward and forward.

STRICTURE.—Always traumatic.

3. SPONGY URETHRA.

LENGTH.—Six inches or more.

SHAPE.—Horizontal slit, except at meatus, where it is a vertical slit.

DIAMETER.—¼ in. No. 12 sound fills it without tension. Narrowest point of whole canal is at the external meatus.

STRUCTURE.

Mucous membrane, with columnar cells. Pits or lacunæ open into it. The lacuna magna is larger than the rest, and opens into the upper wall far back near the beginning. Minute simple glands—the glands of Littré also open into it.

Very vascular submucous layer underlies the mucous membrane.

Unstriped muscle is round this, and

Erectile tissue of corpus spongiosium surrounds the whole.

DIRECTION.—First horizontal. Then upwards and forwards. Then in the line of the penis.

STRICTURE.—Inflammatory.

Varieties.—(1) SPASMODIC AND CONGESTIVE; (2) INFLAMMATORY; (3) TRAUMATIC.

Spasmodic and Congestive Strictures.

SITUATION.—In the bulbous urethra.

CAUSES.—Acute urethritis. Slight blows on the perineum. Cold and wet. Alcoholic or sexual excess, especially when some organic stricture co-exists. Injury from passage of instruments. Operations on the rectum, perineum, or testis.

TREATMENT.—Hot bath. Hot enema. Morphia and belladonna suppository. If retention is unrelieved, large soft catheter.

Inflammatory Strictures.

AGE.—Commonest in patients from twenty-five to fifty.

CAUSES.—Gonorrhœa—by far the commonest. Ulcer caused by a foreign body. Urethral chancre—very rare.

PATHOLOGY.—The mucous membrane is subject to a suppurative catarrh. At certain spots this penetrates deeply, probably where glands or lacunæ are involved. The mucous membrane here is destroyed and an ulcer produced. The organisms and inflammatory process are thus admitted to the vascular submucous tissue. In the submucous tissue a chronic plastic inflammation occurs, producing a round-celled infiltration. This in time is converted into scar tissue, the contraction of which causes the stricture.

The Stricture.—Mucous membrane is lost by ulceration. Mass of cicatricial fibrous tissue occupies the rest of the urethral wall and penetrates into the spongy body to a greater or less extent.

Urethra Above the Stricture.—Dilated. Septic inflammation, with the loss of the superficial layers of the mucous membrane. Ulcerated in old neglected cases. Orifices of glands and lacunæ dilated. Deep layers of the urethra thickened and indurated.

Bladder.—Well-marked hypertrophy. Dilatation with sacculation occurs much later and less frequently than in prostatic obstruction, owing to youth of the patient. Inflammatory changes occur, with cystitis.

Kidneys and Ureters.—Undergo dilatation. Hydronephrosis. Pyonephrosis and pyelonephritis with advent of sepsis.

POSITION.—Just in front of the triangular ligament is much the commonest place. May be anywhere in the spongy urethra,

NUMBER.—One usually, but multiple occasionally.

VARIETIES.

Annular.—Whole circumference involved, but for only a short length.

Ribbon.—As in last, but for a considerable length of the tube.

Bridled.—Only a part of the circumference affected.

Resilient.—A stricture which contracts rapidly after dilating.

Impassable.—An instrument cannot be passed.

Impermeable.—Urine cannot pass.

SYMPTOMS.

Difficult Micturition.—Stream can be started without delay after straining. Takes a long time to complete the act. Stream is fine and projected with force.

Dribbling after Micturition.—The dilated urethra between the bladder and stricture slowly empties itself and trickles on to patient's clothes.

Irritability of the Bladder.—Frequent micturition.

Retention of Urine.—Acute, ending in extravasation; or chronic, with false incontinence.

Urethral Discharge of a mucopurulent, thin character.

Urine at First shreddy, with small casts from the mucous glands and urethra, passed with the first portion of urine.

Later, mucopurulent and ammoniacal from the changes of cystitis.

Cystitis supervenes sooner or later in untreated cases.

PHYSICAL SIGNS.—Those of retention of urine. Obstruction to the passage of a urethral sound or catheter. Induration round the stricture felt externally, especially in bad strictures in the bulb.

Inflammatory Strictures, *continued.*

METHOD OF PASSING INSTRUMENTS.

PRELIMINARY.—Patient lying on his back with head and knees raised. Cleanse the glans and meatus. Syringe the urethra with dilute antiseptic. Inject one drachm of carbolized oil.

TO ASCERTAIN THE EXISTENCE OF AN ORGANIC STRICTURE.—Pass a No. 9 Lister's bougie. If this passes easily, the stricture does not exist or is merely spasmodic.

TO FIND THE POSITION OF THE STRICTURE.—Note the point at which the point of the bougie is arrested. Note the distance from the meatus.

TO FIND THE SIZE OF THE STRICTURE.—Pass small filiform or olive-headed bougies. Note the size of the one which is firmly grasped by the stricture.

TO FIND THE LENGTH OF THE STRICTURE.—Pass acorn-headed bougie through the stricture. Withdraw it and note the distance it is withdrawn, from the moment it engages the stricture until it is released.

Use soft flexible bougies à boule whenever possible. Never use any force in trying to pass a stricture.

DIRECTION.—Pass the instrument with its handle parallel to Poupart's ligament. Then rotate the penis into mid-line. Raise the handle at right angles to the body, and allow the point to slip round the pubic arch.

POINTS WHERE SMALL INSTRUMENTS MAY BE CAUGHT.

In one of the lacunæ, especially the lacuna magna on roof of the urethra. Keep point on the floor of the urethra as far as perineum.

At the triangular ligament. Keep the point on the roof of the urethra as it passes round the pubes.

In the sinus pocularis in floor of prostatic urethra. Keep the point on the roof of the prostatic urethra.

INJURIES WHICH MAY RESULT FROM PASSAGE OF INSTRUMENTS.

HÆMORRHAGE indicates abrasion and laceration.

FALSE PASSAGES.—Sharp point of a metal instrument may pierce the urethral wall and re-enter the urethra or bladder, or produce a sinus. Cause great pain and some bleeding. Results: Increased symptoms of retention—Peri-urethral abscess—Perineal fistula.

CYSTITIS or EPIDIDYMITIS from sepsis.

URINARY FEVER.—Sapræmia due to absorption of toxins through abrasion. Septicæmia due to invasion of the kidneys by micro-organisms.

SHOCK may occur in nervous patients.

SUPPRESSION OF URINE is rare, and supervenes on some previous disease of the kidneys.

Traumatic Stricture of the Urethra.

CAUSE.—Follows rupture of the membranous urethra.

POSITION.—Usually at the (superficial) triangular ligament.

CHARACTER.—Dense mass of scar-like cartilage extending on both sides of the triangular ligament. Sound grates upon its surface. It rapidly contracts, even if it can be dilated.

RESULTS.—Acute or chronic retention. Dilatation and ulceration of the urethra behind the stricture. Perineal abscess or fistula. Extravasation of urine.

Treatment of Organic Strictures.

I. OF PASSABLE STRICTURES.

1. GRADUAL DILATATION.—Used for the majority of cases. Use Lister's bougies. Pass two or three sizes once a week. Continue until a No. 9 to 12 passes easily.
2. CONTINUOUS DILATATION.—Useful in a small stricture with severe retention. Tie in a small instrument for forty-eight hours. Then a larger size for the same time. Later practise gradual dilatation.
3. EXCISION OF THE STRICTURES.—Suitable for cases in which catheterization is badly borne. Cases which rapidly relapse after dilatation. Very dense strictures. Stricture of one inch or less in length.

   METHOD.—Pass a sound or Syme's staff down to or through the stricture. Expose the urethra and split it through the stricture. Cut out the stenosed part. Sew together the two healthy ends. Unite the longitudinal incision through the urethra round a soft No. 10 catheter. Drain the external wound. Tie the catheter in.
4. INTERNAL URETHROTOMY.—Very inferior to excision. Used in the same class of cases. Limited to those which admit a No. 4 bougie.

   METHOD.—Civiale's urethrotome passed through the the stricture. The knife is unsheathed. The instrument withdrawn. The knife is sheathed after cutting through the stricture. In the bulbous urethra cut towards the roof. In the penile urethra cut towards the floor. Tie in a No. 10 catheter for some days.
5. EXTERNAL URETHROTOMY.—Same class of cases as excision. The stricture must admit a No. 2 bougie.

   METHOD.—Pass a Syme's staff with the small end through the stricture. Cut through the stricture from behind forwards. Tie a catheter in the bladder. Leave the perineal wound open.

II. OF IMPASSABLE STRICTURES.

1. WITHOUT RETENTION.

   WHEELHOUSE'S OPERATION.—Pass a Wheelhouse's staff. Open the urethra upon the groove. Retract the opening by the hook on the staff. Cut open the stricture from before backwards. Tie in catheter.

   EXCISION OF THE STRICTURE should always be attempted as a final stage where this is practicable.

Treatment of Organic Strictures, *continued.*

2. WITH RETENTION.
   - COCK'S OPERATION.—Median perineal incision. Stab incision into the prostatic urethra, using a finger in the rectum as guide.
   - URETHROTOMY ON A MORE DELIBERATE PLAN IS MUCH BETTER.—Dissect down to the urethra on the proximal side of the stricture. Open the urethra and pass in a catheter. An excision may often be done later, when the patient and the local conditions are more healthy.

Local Complications.

1. PERINEAL ABSCESS.—Caused by ulceration of the urethra behind the stricture. Septic urine leaks into peri-urethral structures.
   - TREATMENT.—Open freely externally. Find the opening into the urethra. Pass a full-sized catheter into the bladder. Treat the stricture later.
2. PERINEAL FISTULÆ.—The result of an untreated abscess. Generally multiple, opening on to the perineum, buttocks, or scrotum. Lead into tortuous sinuses buried in dense cicatricial tissue.
   - TREATMENT.—Lay all the sinuses freely open. Open the urethra above the stricture. Tie in a catheter from bladder to perineum. Treat the stricture later or at the same time.
3. EXTRAVASATION OF URINE.

EXTRAVASATION OF URINE.

Causes.

RUPTURE OF THE BLADDER—extra-peritoneal.

RUPTURE OF THE URETHRA behind a stricture.

1. DIRECT RUPTURE following acute retention.
2. RUPTURE OF AN ULCERATED URETHRA following chronic retention.

RUPTURE OF A PERI-URETHRAL ABSCESS into the subcutaneous tissues.

INJURIES TO THE URETHRA by instruments which have forced false passages round the stricture and re-entered the urethra above the stricture.

TRAUMATIC RUPTURE of the urethra.

Anatomy.

MEMBRANOUS URETHRA is ruptured usually at or near the superficial triangular ligament.

URINE escapes through the triangular ligament.

- IS DIRECTED BY COLLES'S FASCIA: Forwards to the scrotum and penis; upwards to the anterior abdominal wall.
- IS BOUNDED BY ATTACHMENT OF THE SUPERFICIAL FASCIA to inner end of Poupart's ligament, and thence horizontally across the thigh to the fascia lata.

Symptoms.—Sensation of rupture when straining to relieve retention. Feeling of relief of the retention without passing urine. Rapidly fatal sapræmia or septicæmia.

Physical Signs.—Rapidly increasing boggy swelling of the perineum, external genitals, anterior abdominal wall. Parts becoming dusky and then gangrenous. An emphysematous crackling is caused by the gas formed in the subcutaneous tissues. Extensive sloughing of the skin occurs if the patient recovers.

Treatment.—Early free incisions into all the affected parts. Open the urethra above or through the stricture. Tie in a catheter from the bladder to the perineum. Hot hip baths, with fomentations over all the incisions.

CHAPTER XLVII.

DISEASES OF THE MALE GENITAL ORGANS.

DISEASES OF THE PENIS.

Malformations.

PHIMOSIS and PARAPHIMOSIS (*vide infra*).

EPISPADIAS.—Urethra opens on the dorsum of penis. Involves a torsion of the penis. Is usually associated with ectopia vesicæ.

TREATMENT by plastic operation in suitable cases.

HYPOSPADIAS.—Urethra opens on the lower aspect of the penis or in the perineum. There are three grades: (1) Meatus opens below the glans; (2) Meatus opens below the penile body; (3) Meatus opens in the perineum. The last is associated with cleft scrotum and defective development of penis and testis.

TREATMENT by plastic operations in the more severe cases.

TORSION OF THE PENIS.—The organ is twisted. Usually combined with epispadias.

DISLOCATION (very rare).—The whole organ is pulled out of its cutaneous coverings, and lies under the skin of the scrotum or groin.

DOUBLE PENIS.—(Very rare.)

Phimosis.

1. CONGENITAL.—From a long, narrow prepuce. From a minute preputial orifice. From adhesion of the prepuce to the glans.
   - SYMPTOMS.—Painful and difficult micturition. Ballooning of the urethra. Dribbling of urine after micturition.
   - RESULTS.—Eczema of and round the genitals. Stone in the bladder. Cystitis. Dilatation of bladder, ureters, and kidneys. Hernia. Prolapsus recti.
2. ACQUIRED.—From the contraction of fissures at the orifice. From any inflammatory affection of the prepuce.
   - RESULTS.—Paraphimosis. Rupture of the frænum. Liability to severe forms of venereal disease, or epithelioma.

TREATMENT.

1. CIRCUMCISION.—In all uncomplicated cases.
   - METHOD.—Cut off the prepuce after pulling it forward over the glans. Cut the mucous membrane of the prepuce near to the glans. Sew the edges of skin and mucous membrane with catgut.

ERRORS.—Removal of too much skin: Retracts the penis—Prevents erection—Liable to ulceration. Removal of too little skin: The phimosis recurs.

2. SLITTING UP THE PREPUCE along its dorsal aspect. In cases in which a chancre or some foul secretion is concealed by the prepuce, or in which paraphimosis has occurred.

Paraphimosis.

CAUSED BY constriction of the glans at the corona by a contracted preputial orifice after the prepuce has been drawn back.

SIGN AND RESULTS.—Pain and œdema. Ulceration at the constricted point. Gangrene of parts below the constriction.

TREATMENT.—Reduction under an anæsthetic. Division of the prepuce along mid-dorsal line if reduction is impossible.

Circumcision—At the time if the parts are healthy. Later if much inflammation exists.

Priapism.—Continuous erection in the absence of sexual desire.

CAUSES.—Venereal excess. Injury or thrombosis in corpora cavernosa. Phimosis or stone in the bladder (children). Leukæmia. Injuries to the cervical spinal cord.

SYMPTOMS.—Pain. Mental distress. In nerve cases there is turgescence without any pain or rigidity.

TREATMENT.—In nerve cases leave it alone.

SEDATIVES: bromides, antimony, morphia, etc.

INCISIONS into the corpora cavernosa.

Tumours.

SEBACEOUS CYSTS.

HORNS.—Growing from a ruptured sebaceous cyst.

PAPILLOMA.—Common in gonorrhœa. Usually grow inside the prepuce, or from the glans. Are soft, friable, dendritic masses. There is no infiltration of their base.

Treated by removal with the scissors, touching the base with the cautery. Or by application of calomel.

EPITHELIOMA (*vide infra*).

SARCOMA (very rare).

Epithelioma.

CAUSES.—Men over 40. Almost always occurs in conditions of phimosis. Unknown among Jews. Any ulcer, wart, or fissure may give rise to it.

POSITION.—From the deep surface of the prepuce. Glans.

CHARACTER.—(1) Cauliflower-like mass of bleeding warts with indurated base; or (2) A deep ulcer with indurated base and edges, often concealed by the phimosis and producing a foul sanious discharge.

PROGRESS.—May form an advancing fungating mass or a deep, foul ulcer. Either kind may slough through the prepuce. The urethra is often opened behind the meatus, causing a urinary fistula. Inguinal glands enlarge, and often suppurate. The lymphatics may stand out as red lines. A solid œdema may result from lymph obstruction.

Epithelioma of the Penis, *continued.*

DIAGNOSIS.

PAPILLOMATA.—Have no induration.

CHANCRE, or any indurated ulcer with phimosis. If it does not quickly yield to treatment, slit up the prepuce, remove and examine.

TERTIARY ULCERATION (so-called relapsing chancre).—Follows a primary chancre. Yields to the action of iodides. Is avascular. There is little or no new growth at the edges.

TREATMENT.

AMPUTATION through the body of the penis. In early cases confined to the glans.

COMPLETE REMOVAL OF ALL GLANDS from both groins.

AMPUTATION OF THE WHOLE PENIS: In advanced cases. The scrotum is split. The crura are dissected away from the pubes.

DISEASES OF THE TESTIS.

Congenital Abnormalities.

POLYORCHISM (more than two testes), ANORCHISM (no testis), and MONORCHISM (one testis), are all very rare.

RETAINED TESTIS.—The testis remaining in the abdomen or inguinal canal is common.

DEGREES OF RETENTION.

Abdominal—It is attached to the posterior abdominal wall.
Iliac—It lies deep to the internal abdominal ring.
Inguinal—It lies in the inguinal canal.

STRUCTURE AND SIGNS.—A soft or tender lump exists at the internal ring or in the canal. The scrotum is ill-developed, and empty on that side. The testis is usually soft, ill-developed, and functionless.

COMPLICATIONS.—Hernia usually co-exists. Complete atrophy of the testis. Torsion and gangrene. Orchitis. Hydrocele or hæmatocele. Malignant disease.

DIAGNOSIS from: Hernia, hydrocele of the cord, and other inguinal swellings.

The absence of the testis from the scrotum is the main clue to the diagnosis.

TREATMENT.—None required except for complications. If a hernia has to be operated upon, a retained testis, being functionless, is best removed.

ECTOPIA TESTIS.—Testis leaves the inguinal canal in an abnormal position. Various positions: Perineal, crural, near the anterior-superior spine. Pubic.

TREATMENT.—Removal if it is painful or inflamed.

TORSION OF THE SPERMATIC CORD.—The testis is rotated once or twice, and the cord is twisted. The twisted cord causes strangulation of the vessels. It probably arises from conditions of imperfect descent and development.

SYMPTOMS.—Sudden sickening pain in the groin. Great swelling of testis and of the cord below strangulation. Vomiting and collapse.

DIAGNOSIS.—From strangulated hernia. By the following points: Any evidence of non-descent of the testis. The epididymis being in front of the testis. A normal part of the cord above the swelling. Absence of constipation.

ANATOMY.—Great swelling and ecchymosis of the epididymis. The body of the testis is comparatively normal. Possible gangrene of the whole organ.

TREATMENT.—Removal of the testis and cord. Replacement is seldom desirable or justifiable.

HYDROCELE.

Definition.—A collection of serous or spermatic fluid connected with the testis, cord, or their coverings.

Varieties.

I. VAGINAL, i.e., in a normal or abnormal tunica vaginalis.
  1. CONGENITAL.—In the whole funicular and vaginal tunics, whose cavity communicates with the peritoneum.
  2. INFANTILE.—Similar to the congenital, but shut off from the peritoneum at the internal ring.
  3. BILOCULAR.—In an abnormal, bilocular, funicular sac.
  4. ACQUIRED:
    (*a*) Primary.—A chronic affection of the tunica vaginalis itself.
    (*b*) Secondary.—May be acute or chronic, secondary to disease or injury of the testis or epididymis.

II. HYDROCELE OF THE CORD, usually funicular.
  1. ENCYSTED HYDROCELE OF THE CORD.—Arises in an unobliterated part of the funicular process shut off from the tunica vaginalis below.
  2. FUNICULAR HYDROCELE.—Arises in the patent funicular process.
    (*a*) Congenital.—Communicates with the peritoneum.
    (*b*) Infantile.—Is shut off from the peritoneal cavity.
  3. DIFFUSE HYDROCELE OF THE CORD. — A condition of lymphatic œdema.

III. HYDROCELE OF THE TESTIS AND EPIDIDYMIS.
  1. ENCYSTED HYDROCELE OF THE EPIDIDYMIS, from a dilatation in the spermatic ducts.
  2. ENCYSTED HYDROCELE OF THE TESTIS.—Arises beneath the tunica albuginea.

IV. HYDROCELE OF A HERNIAL SAC.—In infancy this will be a congenital hydrocele. In adults, a similar condition may arise in the sac of a hernia after its contents have been reduced.

Hydrocele, *continued.*

Primary Chronic Vaginal Hydrocele.

CAUSES.—Practically unknown. Probably some inflammatory thickening of the tunica vaginalis.

STRUCTURE.—Tunica vaginalis becomes thickened in proportion to the age of the hydrocele. It may become fibrous, cartilaginous, or calcified. Fibrous or warty growths may be found free in the cavity, or growing from the surface of the tunic.

The fluid is usually about 4 to 12 oz., sp. gr. 1025, neutral reaction, 6 per cent albumin. Coagulates when mixed with a little blood. It is usually clear straw-coloured. It may be green (blood pigments), turbid (mucous), or shimmering (cholesterin crystals).

ANATOMY.—The enlarged tunic surrounds the testis, which lies at the back and lower part of the swelling. The enlargement distends the scrotum, and has but little or no tendency to travel up the cord.

SIGNS AND SYMPTOMS.—A scrotal swelling with the following characters: Large size, but of slow formation. Absence of much pain or tenderness. Feels smooth on the surface, is globular or pyriform. Elastic or fluctuating in proportion to thinness and tenseness of the sac.

Translucency.—This may be absent: When the fluid contains much blood or mucus; if the tunica vaginalis is very thick; if many adhesions exist inside the sac.

It is dull to percussion. There is no impulse on coughing. It is bilateral in about one case in five.

DIAGNOSIS.

HERNIA.—In this there is: A swelling which extends into the inguinal region. Impulse, reducibility, gurgling, resonance. An irreducible epiplocele is the most difficult to distinguish. The obscurity of the cord, the doughy feel, and the opacity, are the chief points. The two conditions may co-exist.

HÆMATOCELE: Feels harder, heavier, and less elastic. Is not translucent. Often gives a history of injury. There may be some ecchymosis.

SOLID ENLARGEMENTS OF THE TESTIS AND EPIDIDYMIS.—These are opaque and do not fluctuate. The outline of the body and epididymis is usually to be felt. Pain and tenderness are generally marked. The cord is often thickened. In inflammatory enlargements of the organ a secondary hydrocele may be present, but inflammatory signs are evident.

A sarcoma is the most difficult to distinguish, because it is often so elastic.

OTHER VARIETIES OF HYDROCELE.—Hydroceles of the cord and epididymis and testis leave the body of the testis unobscured. Congenital and infantile hydrocele run up to the internal ring, the congenital variety being reducible.

TREATMENT.

Tapping.—Make sure of the position of the testis. Drive the trocar upwards and backwards so as to clear the testis. It has to be repeated every few months.

Injections of strong antiseptics after tapping. Linimentum iodi, ʒij, water, ℥iv; or pure carbolic and glycerin, āā ʒj.

Excision of part or whole of the sac. Especially indicated when the fluid reaccumulates very quickly after tapping, or when the sac is thick.

Secondary Chronic Hydrocele.—Of small size. Merely obscuring the testicular outline. Secondary to syphilis, tubercle, or chronic inflammation.

Treatment is that of the primary disease. Firm strapping may be useful.

Acute Hydrocele.—Always secondary to some injury or disease of the testis. Gonorrhœa, traumatic orchitis, or wounds may cause it. Usually is absorbed spontaneously, leaving adhesions. Very rarely suppurates.

Treatment is that of the primary disease.

Congenital and Infantile Hydrocele.—Translucency is well marked. The swelling extends into the groin. Congenital hydrocele is slowly reducible.

Treatment.—Usually disappear spontaneously. Tapping (in infantile), followed by a truss. Injections are dangerous. Operate only if a hernia co-exists.

Bilocular Hydrocele.—A form of infantile hydrocele. The open funicular process has a secondary pouch opening from it. This may lie between the peritoneum and muscles, between the muscles, or between the skin and muscles.

Treatment.—By open incision, dissecting out as much as possible.

Hydrocele of the Cord.—A fluid swelling above and separated from the testis. Congenital form is reducible into the peritoneum. Infantile form extends into the inguinal canal, but is irreducible. Encysted form is a globular enlargement at one part of the cord. It may be multiple. Commoner on right side than on left.

In addition to translucency and fluctuation, all these are distinguished by moving with traction on the testis. And usually an upper limit can be defined, so distinguishing them from herniæ.

Treatment.—Acupuncture or excision.

Encysted Hydrocele of the Epididymis.

Formed by a Dilatation of one of the Ducts, or fœtal tubes, at the head of the epididymis.

Usually Contains Altered Spermatic Fluid. It is alkaline, with little or no albumin. Opaque, milky appearance. It effervesces with acetic acid. It contains many living or degenerate spermatozoa.

Encysted Hydrocele of the Epididymis, *continued.*

It forms a swelling ABOVE AND DISTINCT FROM THE TESTIS and tunica vaginalis.

Rarely contains more than a few ounces of fluid.

Encysted Hydrocele of the Testis.—Occurs beneath the tunica albuginea. Usually of quite small size. Resembles the last variety when it becomes large.

TREAT both this and the last by acupuncture or excision.

HÆMATOCELE.

Definition.—A blood collection in the tunica vaginalis or cord.

Varieties.—Vaginal. The only common variety. Diffuse in the cord. From a ruptured vessel. Encysted in the testis, epididymis, or cord. Arises from bleeding into the corresponding hydrocele.

Cause.—Contusion of the testis. Contusion or puncture of a hydrocele. Secondary to malignant disease.

Structure.—Massive thickening of the walls of the tunica vaginalis, with patches of cartilage or calcareous material. Filled with old and laminated clot, with, perhaps, a little fluid. The testis is often degenerated.

Signs and Symptoms.—An oval tumour, which envelops both testis and epididymis. It is not translucent, and feels solid or doughy. There is history of either a trauma or a hydrocele. Ecchymosis may be well marked in recent cases. The cord is not thickened. The size is stationary, or nearly so.

Diagnosis.

FROM HYDROCELE: This is fluctuating and translucent. Puncture yields clear fluid.

FROM TRAUMATIC ORCHITIS: Diagnosis is very difficult. In this the outline of testis and epididymis can often be made out. Tenderness is much more marked.

FROM SARCOMA, GUMMA, OR OTHER NEW GROWTH: These steadily increase in size. In their early stages the testis and epididymis can be distinguished.

In all doubtful cases it is better to explore.

Treatment.—Palliative in the acute cases after trauma—rest, elevation, and lotions. Operative in chronic cases. Turn out the clot, and excise the sac. Castration in old men with degenerate testis and much pain.

INFLAMMATORY DISEASES OF THE TESTIS AND EPIDIDYMIS.

Varieties.

EPIDIDYMITIS.

ACUTE OR CHRONIC. (1) Simple. (2) Pyogenic. (3) Gonorrhœal. (4) Tuberculous. (5) Syphilitic.

ORCHITIS.

Acute. (1) Traumatic. (2) Complicating a specific fever.

Chronic. (1) Resulting from the acute form. (2) Syphilitic. (3) Tuberculous.

EPIDIDYMO-ORCHITIS.—Begins as an orchitis. Usually traumatic.

Acute Epididymitis.

CAUSES.—Some primary inflammation of the posterior urethra, prostate, or bladder, viz.: Gonorrhœa—Gouty Urethritis—Septic catheterization—Stricture—Prostatitis—Cystitis.

ANATOMY.—The epididymis is much swollen, the tail being chiefly affected. The vas is generally swollen. The tunica vaginalis may be inflamed, and its fluid increased to form an acute hydrocele.

SIGNS AND SYMPTOMS.—A boat-shaped swelling behind the testis, which is very tender, and most marked at the lower end. The scrotum is red, inflamed, and often adherent. There may be a fluid swelling obscuring the testis, from affection of the tunica vaginalis. The vas is swollen, tender, and painful. General malaise, with rise of temperature. Extreme tenderness. Walking with the legs abducted. Acute stage lasts one or two weeks. Often leaves a nodular thickening in the globus minor. The urethral discharge generally diminishes in acute stage. One side is affected after the other.

SEQUELÆ.—Chronic epididymitis. Abscess (very rare). Sterility in bilateral cases.

TREATMENT.—Rest in bed, with support to the scrotum. Evaporating lotions. Hot fomentations if great pain exists. Do not give any urethral injections.

Chronic Epididymitis.

VARIETIES. — Simple, commonly gonorrhœal. Tuberculous, syphilitic (*vide infra*).

CAUSES.—The same as in acute epididymitis. Generally is the remains of an acute attack. Sometimes is chronic from the first.

SIGNS.—An induration and swelling of the epididymis and vas. Most marked at the tail, i.e., the globus minor. May affect the entire epididymis.

SYMPTOM.—Dragging pain in the testis.

RESULT.—Often produces sterility if bilateral.

TREATMENT.—Strapping and suspension of the testis.

Traumatic Orchitis.—Often affects both epididymis and testis. Results from crushes and contusions. Consists of hæmorrhagic and fibrinous masses in the substance of the testis and epididymis. The organ is enlarged, very hard, nodular, and tender. Usually leaves a chronic nodular thickening. May result in atrophy.

TREAT by strapping and suspension.

ORCHITIS.

Whereas epididymitis seldom spreads to the body of the testis, inflammation of the body often spreads to the epididymis.

Acute Orchitis.

CAUSES.—Injury (*vide supra*). Infective fevers: Mumps—Enteric—Variola — Tonsillitis — Scarlatina — Malaria — Influenza—Rheumatism—Gout.

SIGNS AND SYMPTOMS (of the only common form, viz., that in mumps).—Occurs in young adults. Begins about a week after the parotitis. Testis swells to two or three times its natural size. It retains its shape, and is firm and elastic. It is intensely tender and painful. One side may be affected after the other. High fever and prostration occur for a few days, but soon subside. Suppuration almost never occurs in parotitic forms, but may in variola, enteric, rheumatism, or gout. Atrophy is very rare as a sequel.

TREATMENT.—Constitutional treatment, rest in bed, etc. Cooling lotions in most cases. Fomentations in gout or rheumatism.

SYPHILIS OF THE TESTIS.

Varieties. (1) Secondary (intermediate). (2) Tertiary. Interstitial, or gummatous. (3) Congenital.

SECONDARY SYPHILIS of the testis.—Occurs in the late secondary stage—" Reminders." Often bilateral. Affects the epididymis, and especially the globus major. Often associated with small hydrocele. May leave a fibrous nodule in the globus major.

TREAT with mercury.

Tertiary Syphilis of the testis.

PATHOLOGY.—Always affects the body of the testis primarily. Two distinct processes may occur, viz., interstitial orchitis, and gumma. Or both these may be present in the same case. The interstitial change consists in a massive fibrous thickening of the trabeculæ of the gland, which compress and destroy it. The gumma produces a localized caseating mass, which soon reaches the surface, adheres to the scrotum, and breaks through it, forming an ulcer or fungus testis. The tunica vaginalis is dilated or obliterated.

SIGNS.—Slow, steady enlargement of the body of the testis. Mass is nodular, heavy, painless, and without testicular sensation. A hydrocele often co-exists. When gummata occur, a localized fluctuating swelling is felt, followed by an ulcer or fungus. It often affects one testis after the other. It leads to atrophy unless treated early.

TREATMENT.—Iodides with mercury. Strapping with mercury plaster.

Congenital Syphilitic Disease of the testis occurs in infancy or childhood. Usually bilateral. It is similar to the acquired form. The testes are large, very hard, and nodular.

Almost impossible to distinguish from tuberculous disease in children, except by disease of other parts. Tuberculous disease is generally unilateral.

TUBERCULOUS DISEASE OF THE TESTIS.

Etiology.—Occurs in young adults, or even in young children. May be a primary tuberculous lesion, or more often is secondary to disease elsewhere, especially bladder, prostate, vesiculæ, kidneys, or lungs.

PREDISPOSING CAUSES: Any injury of the testis, especially gonorrhœa or traumatic orchitis.

Pathology.

EPIDIDYMIS is affected first almost invariably.

THE GLOBUS MAJOR is the earliest seat of the disease.

NODULAR INFILTRATION, caseation, and fistula formation succeed each other. In chronic or cured cases, and especially in children, a hard fibrous mass of scar tissue is left permanently.

THE DISEASE MAY EXTEND into the following tissues: Vas deferens, vesiculæ seminales, prostate, tunica vaginalis (producing hydrocele, or obliteration by adhesions), scrotum (forming a fistula or fungus), the body of the testis (rarely), the bladder and kidneys.

Signs.—EPIDIDYMIS SHOWS NODULAR THICKENING, first about the head, then the tail, then the body. These are hard and not inflamed at first. After several weeks, or months, softened areas are felt.

THE SKIN OF THE SCROTUM becomes red and adherent, and the abscess breaks, discharging tuberculous pus.

A FISTULA or FUNGUS TESTIS results.

The tunica vaginalis at first is filled by a small hydrocele. Later it is usually obliterated by adhesions.

The fungus, or fistula, may open behind without involving the tunic, or it may first invade the tunic, and then open in front.

The opposite testis generally is involved later on, and will present an earlier stage of the same disease.

THE VAS is felt as a thickened cord.

THE PROSTATE AND SEMINAL VESICLES may be felt to be thickened and nodular per rectum.

Course.—As in tuberculous disease elsewhere the rate of progress varies much. Usually it is a slow process, lasting many months. Death occurs generally by infection of the bladder, kidneys, or lungs. Cases may become chronic, and a curative fibrosis take place. In this case the ducts will almost certainly be obliterated and, therefore, for reproductive purposes the testis is functionles

Tuberculous Disease of the Testis, *continued.*

Treatment.

MEDICAL TREATMENT in chronic or slight cases.

CASTRATION early in the disease, when secondary deposits are absent, in the hope of a radical cure; or late in the disease, for the relief of fistulæ or fungus.

EPIDIDYMECTOMY.—In bilateral cases, in order to preserve the general influence of the glands.

As extensive removal of the vas as possible should always be done.

Diseased vesiculæ seminales may be removed by the perineal route if the bladder and kidneys are sound.

If the bladder and kidneys are affected it is useless to operate, except as a palliative measure for fungus or fistulæ.

NEW GROWTHS OF THE TESTIS.

Varieties.—Adenoma, or fibrocystic disease—Sarcoma—Carcinoma.

Fibrocystic Disease.—Occurs in young adults. It is very chronic in its course, lasting for years. Produces a very large lobulated tumour. Any invasion of the scrotum, or affection of the vas, is very rare. It probably grows from the organ of Giraldès (the remains of the Wolffian body). The body of the testis is spread over the surface of the tumour. The mass consists of a congery of cysts of different size, with marked mucoid and cartilaginous elements. It resembles the endotheliomata of the parotid gland.

TREATMENT.—Removal.

Sarcoma.—Occurs in children under 10, or at 30 to 40.

VARIETIES.—Usually a mixed or spindle-celled sarcoma. A lymphosarcoma is rare. Mucoid and cartilage elements may appear, in which case it is probably an endothelioma.

SIGNS.—Rapid, painless enlargement of the body of the testis. The tumour is firm, smooth, and elastic. It may begin after a blow. Secondary deposits occur in the lumbar glands, and then in other organs. Affection of the vas or scrotum is late. It sometimes affects both sides.

TREATMENT.—Castration.

Carcinoma Testis.—Occurs in men of forty-five or over. Takes the form of a soft medullary growth. Quickly affects the vas and fungates through the scrotum.

TREATMENT.—Castration.

CHAPTER XLVIII.

THE DIAGNOSIS OF SWELLINGS IN THE INGUINO-SCROTAL REGION.

In the region of the groin and scrotum so many diverse swellings may appear that their differentiation is often a matter of difficulty. In order to meet this, it is important to bear in mind, first, the different groups of diseases that may occur, and second, the importance of a systematic examination of the parts. It is particularly desirable that the affected side should be compared with the unaffected, that the patient should be examined, first, standing up, and then lying down, and that he should be placed facing a good light, so that light and shade are symmetrically distributed on both sides.

Classification of Conditions.

1. HERNIA.
    - ANATOMICAL VARIETY.—Inguinal (oblique, direct, congenital, funicular) or femoral.
    - NATURE OF CONTENTS.—Gut, viscus—e.g., ovary or bladder,—omentum, or fluid.
    - PATHOLOGICAL CONDITION.—Irreducibility, obstruction, strangulation.
2. AFFECTIONS OF THE SPERMATIC CORD.
    - HYDROCELE (congenital, encysted, diffuse).
    - SOLID TUMOURS, e.g., lipoma.
    - EXTENSION FROM TESTICULAR SWELLING—malignant or tuberculous.
    - VASCULAR.—Inguinal varix.
3. AFFECTIONS OF THE TESTIS AND ADNEXA.
    - HYDROCELE.—Vaginal, primary, secondary, encysted, etc.
    - INFLAMMATORY.—Acute or chronic orchitis and epididymitis.
    - SPECIFIC.—Syphilitic and tuberculous disease.
    - TRAUMATIC.—Epididymo-orchitis.
    - VARICOCELE.
4. AFFECTIONS OF THE SKIN AND SUBCUTANEOUS TISSUES.
    - Elephantiasis, œdema, extravasation of urine, epithelioma, sebaceous or dermoid cysts, warts, horns, syphilitic or tuberculous eruptions.

Classification of Conditions, *continued.*

5. GLANDULAR.
    - INFLAMMATORY.—Secondary to irritation or sepsis in the leg, genitals, perineum, or abdomen.
    - SPECIFIC.—Tuberculous, syphilitic.
    - NEW GROWTH. — Lymphadenoma, epithelioma, melanotic sarcoma.
6. ABSCESS.
    - GLANDULAR.—From the suppuration of any of the last mentioned.
    - PSOAS OR ILIAC.—Tracking beneath Poupart's ligament.
    - PELVIC (e.g., parametric).—Tracking through the inguinal canal.
    - HIP DISEASE.—In Scarpa's triangle.
7. MISPLACED VISCUS (other than herniæ).
    - RETAINED TESTIS, or ECTOPIA TESTIS, with any of its complications.
    - OVARY in the inguinal or femoral canal.
8. VASCULAR SWELLINGS.
    - ARTERIAL.—Aneurysm of the iliac or femoral arteries.
    - VENOUS.—Varix of the inguinal, testicular, or saphenous veins.
    - LYMPHATIC.—Hygroma.
9. BURSAL.—Psoas bursa over the hip joint.
10. SARCOMA OF BONES.—May grow from any part of the pelvis or femur.

Differentiation of the Different Structures normally present.—The following parts should be systematically examined in order to determine the extent of the disease, and gain information as to its character.

The skin, especially of the scrotum.
The body of the testis.
The epididymis and vas.
The inguinal canal and its contents.
The tunica vaginalis.
The spermatic veins.
The lymph-glands.

The History of the Swelling in relation especially to :—

Time and mode of onset.	Gonorrhœa.
Syphilis.	Trauma.
Tuberculous Disease.	Sepsis.

The Position of the Swelling.

PURELY SCROTAL SWELLINGS.

IN THE TISSUES OF THE SCROTUM.—Gumma, epithelioma, cysts, extension from disease of the testis.

IN THE BODY OF THE TESTIS.—The mass is chiefly solid, and the epididymis cannot be differentiated. Orchitis (especially that associated with parotitis), traumatic lesions, gumma, carcinoma, sarcoma, cystic disease.

- IN THE EPIDIDYMIS.—When the body of the testis can be distinguished from the epididymis the swelling is usually primary in the latter. Epididymitis, tubercle, syphilitic nodules.
- IN THE TUNICA VAGINALIS.—Fluid, fluctuating, generally translucent swellings obscuring the outline of the testis and epididymis. Vaginal hydrocele, primary or secondary; hæmatocele. It is important always to examine the testis after the evacuation of the hydrocele fluid, so as not to overlook a primary disease of that organ.
- ENCYSTED HYDROCELE.—A fluid translucent swelling, distinct from the body of the testis. Encysted hydrocele of the testis, epididymis, or cord.
- VARICOCELE.—A vermiform swelling distinct from the testis and vas.

PURELY INGUINAL SWELLINGS.

- IN THE INGUINAL CANAL.—Hernia, retained testis, hydrocele of the cord, tumours of the cord, inguinal varix.
  - The fact that an inguinal swelling affects the cord is shown by its movement on pulling the testis.
- SUPERFICIAL TO POUPART'S LIGAMENT.—Lymph-glands.
- ILL-DEFINED inflammatory swellings arising in the glands or coming from the pelvis.

CRURAL SWELLINGS.—Femoral hernia, aneurysm, saphenous varix, hip abscess, psoas bursitis.

INGUINO-SCROTAL SWELLINGS are distinguished by the fact that the scrotal swelling is continuous with one which occupies the inguinal canal.

- INGUINAL HERNIÆ.—These descend from above downwards. Usually give an impulse on coughing, are reducible, and may be resonant to percussion.
- HYDROCELE.—A large vaginal hydrocele may spread up the cord. Hydrocele of the cord. Congenital or infantile hydroceles. All are fluctuating and translucent.
- INFLAMMATORY, TUBERCULOUS, and MALIGNANT swellings spread up from the epididymis to the cord.
- SPERMATIC VARICOCELE may be associated with one in the inguinal region.

SWELLINGS WITH INDEFINITE OR EXTENSIVE LIMITS.

- PSOAS OR ILIAC ABSCESS.
- CYSTIC HYGROMA.
- SARCOMA OF BONES.
- EXTRAVASATION OF URINE.

Examination of Associated Structures.—This often gives the clue to the nature of the disease in obscure cases. The most important structures to be examined are :—

- THE PENIS AND URETHRA, for evidence of syphilis or gonorrhœa.
- THE SKIN of the abdomen, legs, genitals, perineum, and anus, for septic or malignant disease.
- THE SPINE AND HIP, for evidence of tuberculous disease.
- THE PROSTATE AND VESICULÆ SEMINALES, for any thickening or tenderness, which may be due to inflammatory or tuberculous disease.

Physical Examination of the Swelling.

PHYSICAL CHARACTERS ON PALPATION.

SOLID SWELLINGS are those of the testis and epididymis, whether inflammatory, new growth, tubercle, or syphilis. Glandular masses, septic, syphilitic, tuberculous, or new growth. Bony sarcomata.

FLUCTUATING SWELLINGS are hydroceles, hæmatoceles, abscesses, cysts, and gummata.

ELASTIC SWELLINGS, difficult to distinguish from fluctuating, may be sarcoma or gumma of the testis.

DOUGHY.—Herniæ, especially epiploçeles.

HARD, HEAVY, AND INDOLENT, especially diffuse gummata of testis and some forms of new growth.

TENDER AND INFLAMED.—Epididymitis, bubo, and other septic glandular swellings, torsion of the testis, inflamed or strangulated hernia, any acute abscess.

REDUCIBILITY.

THE POSSIBILITY OF REDUCTION into the abdomen proves the swelling to be a hernia, varicocele, saphenous varix, or congenital hydrocele.

THE DIRECTION OF REDUCTION may help to distinguish between an oblique inguinal hernia, which goes upwards and outwards; a direct inguinal hernia, which goes backwards; and a femoral hernia, which goes downwards and then upwards.

THE MANNER OF REDUCTION.—A hernia slips back suddenly, and that containing gut usually gurgles. A congenital hydrocele is reducible very slowly and gradually. A venous swelling is more easily reduced by the patient lying down than by pressure.

THE RECURRENCE AFTER REDUCTION is from above downwards in the case of a hernia, but from below upwards in the case of a varix or congenital hydrocele.

TRANSLUCENCY.—This is well marked in those hydroceles which can be subjected to the test. In old thick hydroceles and in those of the cord it is difficult to obtain.

IMPULSE ON STRAINING.—This is seen in herniæ (if they are not inflamed or irreducible), varicocele, saphenous varix, and some swellings of the cord.

EXPANSILE IMPULSE is peculiar to reducible herniæ, and is rarely seen in congenital hydrocele.

A MERELY PUSHING IMPULSE may be seen in any tumour in the inguinal canal, e.g., a retained testis.

CHAPTER XLIX.

SURFACE MARKINGS.

HEAD AND NECK.

BONY POINTS OF CRANIUM.

NASION.—Naso-frontal suture.

GLABELLA.—Eminence above nasion.

INION.—External occipital protuberance.

BREGMA.—Point where coronal and sagittal sutures meet. The middle of vertical line joining the two pre-auricular points.

LAMBDA.—Junction of lambdoid and sagittal sutures 2½ in. above and in front of inion.

PTERION.—Junction of frontal, parietal, sphenoid, and temporal bones, 1½ in. above centre of zygoma.

SINUSES.

SUPERIOR LONGITUDINAL.—From middle of glabella to inion; linear in front, ¾ in. wide behind. Inclines to the right.

LATERAL AND SIGMOID.—Band ½ in. wide from a point above inion to point ¾ in. behind external auditory meatus, curving so that the highest point of curve is ¾ in. above Reid's base-line.

MENINGEAL ARTERY (middle).

MAIN TRUNK AND ANTERIOR BRANCH.—From middle of zygoma to a point 1½ in. above middle of zygoma (pterion), then upwards and backwards at angle 45°.

POSTERIOR BRANCH.—From the main trunk backwards one finger's breadth above the zygoma.

CEREBRAL HEMISPHERE.—From glabella to pterion, curving downwards and backwards, ½ in. above orbital margin. From pterion to zygoma, curving downwards and forwards. Along zygoma and line of lateral sinus to inion. Mesial border from inion to glabella.

FISSURE OF ROLANDO.—From point ½ in. behind middle of line from nasion to inion. Downwards and forwards for 3⅜ in. at angle 67½° (three-quarters of right angle); (or) downwards and forwards to a point 2 in. above pre-auricular point.

FACE CENTRES.—Lower third of fissure of Rolando.

ARM CENTRES.—Middle third of fissure of Rolando.

LEG CENTRES.—Upper third of fissure of Rolando.

MOTOR SPEECH CENTRE.—(Broca's convolution) just above the left Sylvian point.

Head and Neck, *continued.*

THE SYLVIAN POINT is 1½ in. above the centre of the zygoma, and 1½ in. behind the external angular process It marks: (1) The pterion; (2) The middle meningeal artery; (3) The point of divergence of the anterior, ascending, and posterior limbs of the fissure of Sylvius; (4) The island of Reil; (5) The middle cerebral artery.

THE SYLVIAN FISSURE.—From the Sylvian point to a point ¾ in. below the parietal eminence.

THE OCCIPITO-PARIETAL FISSURE.—A line about 1½ in long drawn outwards towards the external angular process of the frontal bone from a point three-quarters of the way between the glabella and inion.

REID'S BASE-LINE.—From lower margin of orbit to the external auditory meatus to inion.

TREPHINE POINTS.

LATERAL SINUS.—¾ in. behind auditory meatus.

MASTOID ANTRUM.—Suprameatal triangle, or point of junction of tangents, drawn above and behind meatus.

TEMPOROSPHENOIDAL ABSCESS.—¾ in. above post-auricular point.

CEREBELLAR ABSCESS.—1½ in. behind auditory meatus. ¼ in. below Reid's base-line.

LATERAL VENTRICLE.—1½in. above external auditory meatus.

DEAN'S OPERATION.—1¼ in. behind external auditory meatus, ¼ in. above Reid's base line.

SUPRA-ORBITAL NOTCH, INFRA-ORBITAL FORAMEN, AND MENTAL FORAMEN.—Line joining junction of middle and inner thirds of supra-orbital ridge, to interval between two lower bicuspid teeth, crosses each of these.

INFERIOR DENTAL NERVE may be exposed by a trephine midway between the anterior and posterior margins of the ramus on the level of the alveolar border.

FACIAL NERVE.—Crosses ramus of the jaw level with border (lower) of lobule of ear.

PAROTID DUCT.—From lower margin of tragus or concha to point midway between ala nasi and angle of mouth. Ends on margin of masseter opposite second upper molar.

CAROTID ARTERY.—From sternoclavicular joint to midway between angle of jaw and tip of mastoid.

COMMON CAROTID.—Up to the upper border of the thyroid cartilage.

EXTERNAL CAROTID.—Same line above thyroid cartilage.

SUPERIOR THYROID ARTERY, LINGUAL ARTERY, AND FACIAL ARTERY.—Run downwards, forwards, and upwards respectively from a point on the external carotid opposite the great cornu of the hyoid bone.

SUBCLAVIAN ARTERY.—Sternoclavicular joint to middle of clavicle. Rising in curve, which is 1 in. above clavicle.

EXTERNAL JUGULAR VEIN.—From angle of jaw to middle of clavicle.

BRACHIAL PLEXUS.—Upper nerve from a point on the posterior border of the sternomastoid opposite the cricoid cartilage, to a point outside the middle of the clavicle. The lower nerve just above the clavicle.

SPINAL ACCESSORY NERVE.—From point between angle of jaw and mastoid to middle of posterior border of sternomastoid.

UPPER BORDER OF THYROID.—Level of disc between third and fourth vertebræ.

CRICOID CARTILAGE.—Level of fifth cervical vertebra. Omohyoid crosses the carotid sheath. Beginning of œsophagus.

RIMA GLOTTIDIS.—Middle of the anterior border of the thyroid cartilage.

EPIGLOTTIS.—From below the thyroid notch to above the hyoid bone.

ISTHMUS OF THE THYROID GLAND. —A band ½ in. wide, which is ½ in. below the cricoid.

LATERAL LOBE OF THE THYROID extends up to the thyroid cartilage, down to the clavicle, and outwards to the carotid line.

THORACIC VISCERA, Etc.

Parasternal line is a vertical line midway between the edge of the sternum and the midclavicular line.

LUNGS.

APEX.—Extends 1 in. above inner third of clavicle.

MEDIAN BORDER.

RIGHT.—Sternoclavicular joint to middle of manubrium, to seventh costal cartilage in mid-line.

LEFT.—Sternoclavicular joint to middle of manubrium to fourth costal cartilage; along this to parasternal line; down this to sixth costal cartilage.

LOWER BORDER.—Sixth cartilage in parasternal line, to eighth cartilage in midaxillary line, to tenth rib in line of angle of scapula, to tenth dorsal spine (6–8–10.)

FISSURE.—Second dorsal spine downwards and forwards to sixth costal cartilage in parasternal line.

TRANSVERSE FISSURE (on the right side only).—From main fissure forwards along fourth rib and cartilage.

BIFURCATION OF TRACHEA.—In front – the angle between the manubrium and body of the sternum (angulus Ludovici). Behind—between third and fourth dorsal spines.

ROOT OF LUNGS.—Opposite fourth, fifth, and sixth dorsal spine.

Thoracic Viscera, etc., *continued.*

PLEURA.

- APEX.—1 in. above inner third of clavicle.
- MEDIAN BORDER.
  - RIGHT.—From midmanubrium to seventh costal cartilage in mid-line.
  - LEFT.—From midmanubrium to sixth costal cartilage to left of mid-line.
- LOWER BORDER.—From lowest possible point of median border to 2 in. above tip of tenth costal cartilage to twelfth rib, where it is crossed by erector spinæ, to twelfth dorsal spine; or more simply, seventh costal cartilage (parasternal) to ninth rib (midaxilla) to eleventh rib (scapula angle) (7–9–11).

HEART AND PERICARDIUM.—Lie between curved lines joining four points :—

1. Second left intercostal space, upper border 1 in. from sternum.
2. Second right intercostal space, lower border ½ in. from sternum.
3. Sixth right costosternal junction, 1 in. from sternum.
4. Fifth left interspace, ½ in. inside midclavicular line.

- RIGHT AURICULOVENTRICULAR GROOVE.—Joins 1 and 3.
- VALVULAR ORIFICES.—All below and to the left of the line 1–3. In order from above downwards.
- PULMONARY AND AORTIC.—Opposite third costal cartilage.
- MITRAL.—Opposite fourth costal cartilage.
- TRICUSPID.—Opposite fourth and fifth costal cartilages.
- ASCENDING AORTA.—From third left to the second right chondrosternal joint.
- INNOMINATE ARTERY.—From middle of manubrium to right sternoclavicular joint.
- FIRST PORTIONS OF LEFT SUBCLAVIAN AND LEFT CAROTID.—From middle of manubrium to left sternoclavicular joint.
- PULMONARY ARTERY.—From third to second left chondrosternal junction.
- LEFT INNOMINATE VEIN.—Left sternoclavicular joint to apex of the first right intercostal space.
- RIGHT INNOMINATE VEIN.—Right sternoclavicular joint to apex of the first right intercostal space.
- SUPERIOR VENA CAVA.—From first to the third right chondrosternal junction.
- INTERNAL MAMMARY ARTERY.—From sternoclavicular joint vertically downwards to the sixth costal cartilage ½ in. from sternum above, but closer to it below.
- MAMMARY GLAND.—Extends up to the second rib, down to the sixth, inward to the border of the sternum, and outward to the midaxillary line.
  - The nipple lies on the fourth rib, or interspace, outside the midclavicular line.

DIAPHRAGM.—Also highest level of liver (right), stomach (left).

- IN FRONT (5, 5, 5).—Fifth left interspace in midclavicular line.

Junction of ensiform cartilage, to sternum (fifth sternal joint), to fifth right rib in midclavicular line.

BEHIND (8, 8, 8).—Eighth rib in the right scapular line, to eighth spine, to eighth interspace in the left scapular line.

ABDOMEN.

TRANSPYLORIC PLANE, OR PYLORIC LINE.—Horizontal plane midway between upper border of sternum and crest of pubes, and midway between ensiform cartilage and navel.

It also in normal parts is line joining tips of ninth costal cartilage.

It is the level of the following: Pylorus—first part of the duodenum. Gall-bladder—duodeno-jejunal junction. Hila of kidneys—beginning of ureters. Body of pancreas—beginning of mesentery. Body of first lumbar vertebra.

SUBCOSTAL PLANE is on level with third lumbar vertebra.

LINE JOINING HIGHEST POINTS OF ILIAC CRESTS.—Level with fourth lumbar vertebra.

INTERTUBERCULAR PLANE.—Level with fifth lumbar vertebra.

LINEA SEMILUNARIS.—From tip of the ninth costal cartilage to a point midway between the anterior iliac spine and the umbilicus, and thence to the pubic spine.

LINIÆ TRANSVERSÆ.—(1) At the umbilicus; (2) Midway between umbilicus and xiphoid cartilage; (3) At xiphoid cartilage.

INTERNAL ABDOMINAL RING.—Just above middle of Poupart's ligament.

EXTERNAL ABDOMINAL RING.—Just above the pubic crest.

LIVER.

ABOVE IN FRONT.—From left fifth interspace in midclavicular line, to junction between sternum and ensiform cartilage, to fifth rib in the right midclavicular line.

BEHIND.—From eighth interspace in left scapular line, to eighth dorsal spine, to eighth rib in right scapular line.

BELOW.—From left fifth interspace in midclavicular line, to transpyloric line in mid-plane, to right costal margin.

STOMACH.

CARDIAC ORIFICE.—One inch to left of junction of ensiform cartilage.

PYLORIC ORIFICE.—One inch to right of middle of transpyloric line.

HIGHEST LEVEL.—Fifth interspace in left nipple line.

LOWEST LEVEL.—Infracostal line.

DUODENUM.—Begins 1 in. to right of middle of transpyloric line. Ends 1 in. to left of middle of transpyloric line and a little below it. Extends to the right as far as right mid-Poupart line. Extends below as far as line joining highest points of the iliac crests.

Abdomen, *continued.*

MESENTERY.—From 1 in. to left of middle of transpyloric line and a little below it to a point midway between right anterior superior iliac spine and mid-line.

APPENDIX. — Attached end, midway between right anterior superior iliac spine and mid-line.

ILEOCÆCAL VALVE.—Point where right mid-Poupart line crosses intertubercular line.

CÆCUM.—In right iliac fossa below the intertubercular plane, one-third inside and two-thirds outside the mid-Poupart line.

COLON.

BOTH SIDES BEHIND.—Vertical line ½ in. behind centre of iliac crest up to eleventh rib on the right side and to tenth rib on the left side.

ASCENDING COLON (in front).—From the intertubercular plane to the upper border of the ninth costal cartilage, lying outside but adjacent to the right mid-Poupart plane.

TRANSVERSE COLON (in front).—From the ninth right costal cartilage to the eighth left costal cartilage, where these are cut by the mid-Poupart plane. Curving downwards to the infracostal plane in the mid-line.

DESCENDING COLON (in front).—From eighth left costal cartilage to the left iliac crest, outside but adjacent to the mid-Poupart plane.

RECTUM (behind).—From a point 1 in. below the level of the posterior superior iliac spine (which is on a level with the second sacral spine) to the anus.

GALL-BLADDER.—At angle where ninth right costal cartilage is crossed by outer border of right rectus.

PANCREAS.

BODY lies on first lumbar vertebra, level of transpyloric line, and of twelfth dorsal spine.

TAIL from twelfth dorsal spine to the left up to the tenth rib above pyloric line.

HEAD lies below pyloric line level with second lumbar vertebra.

SPLEEN underlies ninth, tenth, and eleventh ribs on left side. Innermost point 1½ in. from tenth dorsal spine. Outermost point left midaxillary line.

KIDNEYS.

BEHIND.—From the level of eleventh dorsal spine to that of third lumbar spine. HILUM.—2 in. from first lumbar spine.

IN FRONT.—Hilum is on transpyloric line 2 in. from middle (tip of the ninth costal cartilage). Thence the kidney extends upwards 2 in. (to the sixth costal cartilage on the left and to the seventh on the right). Outwards 2 in. Downwards 2 in. to the infracostal plane on the left and below this on the right. In each case the upper pole is ½ in. nearer the mid-line than the lower. Left kidney is ½ in. higher than right.

Ureter.—From hilum to posterior superior iliac spine behind. From hilum to bifurcation of iliac arteries in front.

Suprarenal Capsule.—Opposite inner part of eleventh intercostal space.

THE BLOOD-VESSELS.

Abdominal Aorta.—A band 1 in. wide from the tip of the xiphoid cartilage to the line joining the highest points of the iliac crests. Lying to the left of the mid-line.

Inferior Vena Cava.—A band 1 in. wide below and 1½ in. wide above from the line joining the highest points of the iliac crests to the apex of the fifth right intercostal space. Lying to the right of the mid-line. Perforates the diaphragm at its highest point opposite the eighth dorsal spine.

The Cœliac Axis and Superior Mesenteric Arteries arise just above the transpyloric plane.

The Renal Arteries arise just below the transpyloric plane.

The Inferior Mesenteric Arteries arise just above the infracostal plane.

Iliac Artery.—From the left of the middle of line joining highest iliac points to a point midway between the anterior superior iliac spine and the symphysis pubis.

Upper third = common iliac, lower two-thirds = external iliac.

Deep Epigastric Artery.—From a point midway between the anterior superior iliac spine and the symphysis pubis towards the umbilicus.

Deep Circumflex Iliac Artery.—Just above the outer half of Poupart's ligament and round the iliac crest.

The Portal Vein.
The Common Bile-duct.
The Hepatic Artery.
The Foramen of Winslow.

From a point 1 in. to the right of the middle of the transpyloric line upwards and a little to the right to the costal margin. The vein and bile-duct extend 2 in. below the point given; the vein to the left and the duct to the right.

THE SPINAL CORD extends down as far as the first lumbar spine behind, or the transpyloric line in front. The spinal dura mater extends down to ½ in. below the posterior superior iliac spines.

UPPER EXTREMITY.

Acromio-clavicular Joint.—Runs in a sagittal direction midway between the outer and inner borders of the arm when it is hanging at the side. It is just outside a bony tubercle on the anterior margin of the clavicle.

Coracoid Process on the anterior deltoid margin, 1 in. below the junction of the middle and outer thirds of the clavicle.

Upper Extremity, *continued.*

Bicipital Groove—A line 2 in. long downwards from the tip of the acromion in the long axis of the humerus.

Tuberosities.—With the arm everted, the lesser tuberosity lies between the coracoid process and the bicipital groove. The great tuberosity lies outside the bicipital groove.

Head of the Humerus looks in the same direction as the internal epicondyle.

Humeral Epicondyles lie on the same level as the tip of the olecranon when the arm is extended.

Radio-humeral Joint is ¾ in. below the tip of the external epicondyle.

Ulnar-humeral joint is 1 in. below the tip of the internal epicondyle.

The External Intermuscular Septum extends from the insertion of the deltoid to the external epicondyle.

The Internal Intermuscular Septum extends from the insertion of the coracobrachialis to the internal epicondyle.

Axillary and Brachial Arteries.—With the arm abducted and the hand supinated, the line from the centre of the clavicle to the point midway between the humeral condyles and 1 in. below them, represents axillary and brachial arteries. A point one-third of the distance from the outer end of the anterior to the outer end of the posterior axillary folds is the end of the axillary and the beginning of the brachial.

The Radial Artery.—From a point midway between the two epicondyles and 1 in. below them to the radial side of the tendon of the flexor carpi radialis. Thence under the tendons of the extensor ossis metacarpi pollicis and extensor brevis pollicis to the back of the first metacarpal space at its apex.

The Ulnar Artery.—From a point midway between the epicondyles and 1 in. below them to the junction of the upper and middle thirds of a line drawn from the internal epicondyle, to the radial side of the pisiform bone, and thence down this line.

The Superficial Palmar Arch is convex downwards and reaches to a level with the web of the thumb.

The Deep Palmar Arch is one finger's breadth above the superficial.

The Median Basilic and Median Cephalic Veins run from the centre of the antecubital space to the inner and outer borders of the belly of the biceps respectively.

The Basilic Vein runs up the inner border of the biceps and pierces the deep fascia rather more than halfway up the arm.

The Cephalic Vein runs up the outer border of the biceps, and then between the deltoid and pectoral muscles, and pierces the costocoracoid membrane.

The Superficial Lymphatics run with the superficial veins, and end in trunks which accompany the basilic and cephalic veins.

The Lymphatic Glands.

1. At the bend of the elbow—supratrochlear and ante-cubital glands draining the front of the arm and its inner side.
2. Axillary set—lying with the axillary vein on the outer side of the axilla and draining the arm.
3. Pectoral set—along the outer margin of the pectoralis minor, and draining the chest wall and breast.
4. Subscapular group—along the posterior fold of the axilla draining the back, the axilla, and part of the mammary gland.
5. Subclavian group—lying beneath both pectoral muscles below the clavicle, and running up under it to the posterior triangle of the neck. They receive the efferents from all the other groups.

The Circumflex Nerve (and posterior circumflex artery) passes round the back of the humerus just above the centre of the deltoid muscle.

Musculospiral Nerve (and superior profunda artery).—From the junction of the arm and posterior axillary fold to a point one-third of the way from the deltoid insertion, to the external epicondyle, and thence in the groove between the biceps and supinator longus.

Radial Nerve.—From the groove between the biceps tendon and the supinator longus to the junction of the middle and lower thirds of the arm, when it passes beneath the supinator to the back of the wrist.

Median Nerve.—In the arm the same as the brachial artery; at the elbow it lies on its inner side; in the forearm it runs down to the wrist, when it passes beneath the anterior annular ligament to the ulnar side of the flexor carpi radialis.

Ulnar Nerve.—In the arm, at first with the brachial artery; then from the junction of the upper and middle thirds it runs to the groove behind the internal condyle, and thence to the radial side of the pisiform bone.

The Elbow Joint is one finger's breadth below the anterior skin crease.

The Wrist Joint is indicated by the upper of the two skin creases in front.

The Tip of the Radial Styloid Process is in the "anatomical snuffbox," between the tendons of the flexores primi and secundi internodii pollicis. It is on a level with the lower wrist skin crease, ½ in. below the wrist joint.

The Tip of the Ulnar Styloid is just below the level of the upper wrist skin crease, i.e., it is at ½ in. higher level than the radial styloid.

The Tubercle of the Scaphoid is between the tendons of the flexor carpi radialis and the extensor ossis metacarpi pollicis on the lower wrist crease.

The Anterior Annular Ligament is 1 in. wide; its upper border corresponds to the lower wrist skin crease, and it stretches from the pisiform and unciform bones to the scaphoid trapezium.

Upper Extremity, *continued.*

THE COMMON FLEXOR SYNOVIAL SHEATH (which includes the sublimus and profundus tendons and median nerve) extends two or three fingers' breadth above the wrist joint and below to the upper transverse palmar crease. It extends also into the little finger. The flexor longus pollicis has a separate sheath.

THE FLEXOR SHEATHS OF THE THREE MIDDLE FINGERS extend up as far as the lower palmar skin crease.

THE METACARPO-PHALANGEAL JOINTS lie halfway between the lower palmar skin crease and the web of the fingers.

THE INTERPHALANGEAL JOINTS lie between the skin creases of the middle joint and opposite the crease of the lower.

THE TUBERCLE AT THE BACK OF THE RADIUS lies on the outer side of the sheath of the extensor secundi internodii pollicis.

THE TUBERCLE AT THE BACK OF THE CARPUS is the styloid process of the second metacarpal.

THE POSTERIOR ANNULAR LIGAMENT is 1 in. broad, and extends from the back of the radius to the interval between the ulna and the carpus.

THE SYNOVIAL SHEATHS AT THE BACK OF THE WRIST extend about 1 in. above and below the annular ligament. They are :—

1. For the extensor ossis metacarpi and primi internodii pollicis.
2. For the extensores longus and brevis carpi radialis on the floor of the "anatomical snuffbox."
3. For the extensor secundi internodii pollicis outside the radial tubercle.
4. For the extensores indicis and communis digitorum behind the radius.
5. For the extensor minimi digiti between the radius and ulna.
6. For the extensor carpi ulnaris between the head and styloid process of the ulna.

LOWER EXTREMITY.

HIGHEST POINT OF ILIAC CREST.—On a level with the spine and body of fourth lumbar vertebra.

TUBERCULAR POINT.—2½ in. behind the anterior superior spine. Level with fifth lumbar vertebra.

POSTERIOR SUPERIOR SPINE.—Level with second sacral spine. Middle of sacro-iliac joint.

GREAT TROCHANTER.—Tip is crossed by Nélaton's line from anterior superior iliac spine to the ischial tuberosity.

BRYANT'S TRIANGLE.—Patient lying horizontal. Draw a horizontal line up from tip of trochanter. Drop a perpendicular line from the anterior superior spine to meet the first line. Join the anterior superior spine to the tip of trochanter. Horizontal line shows the length of the femoral neck. Vertical line shows degree of rotation.

Tip of trochanter is vertically below the tubercle on iliac crest.

SMALL TROCHANTER.—Is felt above the inner end of the gluteal fold when the femur is internally rotated.

LOWER BORDER OF THE GLUTEUS MAXIMUS.—Join junction of upper and middle thirds of the femur to the middle of the gluteal fold and produce upwards.

GLUTEAL ARTERY.—Leaves the pelvis at a point at the junction of the upper and middle thirds of a line joining the posterior superior spine to the tip of the great trochanter.

SCIATIC AND PUDIC VESSELS.—Leave the pelvis at a point at the junction of the middle and lower thirds of a line joining posterior superior spine to the ischial tuberosity.

GREAT SCIATIC NERVE.—Vertical line midway between the great trochanter and the ischial tuberosity.

EXTERNAL INTERMUSCULAR SEPTUM OF THE THIGH.—Line joining the iliac tubercle to the head of the fibula.

TENDON OF THE BICEPS.—External hamstring.

TENDON OF THE SEMITENDINOSUS.—Internal hamstring.

TENDON OF THE SEMIMEMBRANOSUS.—Lies deep to the internal hamstring and then winds round underneath the inner head of the gastrocnemius; a bursa lying between the two tendons.

HIP-JOINT is marked by a circle inscribed in the following triangle :—

Vertical line dropped from the anterior superior spine. Horizontal line drawn out from the pubic spine, Poupart's ligament.

PUBIC SPINE is felt :—

In the male, by invagination of the scrotum into the external ring.

In the female, by following up the tendon of the adductor longus.

SAPHENOUS OPENING.—1½ in. below and to the outer side of the pubic spine.

FEMORAL ARTERY.—Flex the hip and knee and rotate the thigh outwards. Join the mid-point between the anterior superior iliac spine and the symphysis pubis to the most prominent part of the internal condyle.

Upper two inches represents the common femoral, the rest the superficial. Upper third of line represents the artery in Scarpa's triangle. Middle third of line represents the artery in Hunter's canal. Lower third of line represents the artery in popliteal space.

POPLITEAL ARTERY BEHIND.—From apex of the popliteal space to midway between the two condyles at a point on a level with the tibial tubercle.

ANTERIOR TIBIAL ARTERY.—From a point midway between the head of the fibula and the external tibial tuberosity to a point midway between the malleoli, where it is crossed by the extensor longus hallucis.

Lower Extremity, *continued.*

Dorsalis Pedis.—From midway between the malleoli to the base of the first metatarsal space. It is crossed by the inner tendon of the extensor brevis digitorum.

Posterior Tibial.—From a point midway between the femoral condyles, but on a level with the tibial tubercle, down to a point one finger's breadth behind the inner malleolus.

Internal Plantar Artery.—From the tip of the inner malleolus to the ball of the great toe.

External Plantar Artery.—From the tip of the inner malleolus to the base of the fifth metatarsal bone, and thence to the base of the first metatarsal cleft.

Internal Saphenous Vein and Nerve.—From in front of the inner ankle to the groove between the sartorius insertion and the inner head of the gastrocnemius, behind the inner condyle of the femur and up to the saphenous opening.

External Saphenous Vein and Nerve.—From behind the outer ankle to the middle of the popliteal space.

The Superficial Lymphatics follow the same line as the above two veins.

The Lymphatic Glands.

1. Popliteal group—generally one or two, drain the outer side of the leg or foot.
2. Inguinal group—six to seven, parallel to Poupart's ligament, drain the anterior abdominal wall and upper part of the thigh..
3. Pubic set—two to three, over the pubis, drain the buttocks, anus, perineum, and external genitals.
4. Superficial femoral—two to three, on the inner side of the femoral vessels at the saphenous opening, drain the greater part of the thigh and inner side of the leg and foot.
5. Deep femoral—one or two lie in the crural canal, and drain all the other superficial groups, together with the deep parts of the leg.

Anterior Crural Nerve.—Downwards from a point midway between the anterior superior iliac spine and the spine of the pubes.

Femoral Ring and Crural Canal.—Lie on the outer side of the pubic spine and on the inner side of the femoral vessels.

Adductor Tubercle.—Just above the most prominent part of the internal condyle. In the interval between the vastus internus in front and the sartorius behind. It marks the site of the epiphyseal junction.

Knee-Joint.—In full extension the lower border of the patella is on a level with the joint. In semiflexion a triangle exists in front of the inner side bounded by the inner border of the patella, anterior lower border of the internal femoral condyle, upper border of the internal tibial tuberosity In this a displaced internal semilunar cartilage, or loose body, can be felt.

The Head of the Fibula is on the same level as the tubercle of the tibia.

Internal Lateral Ligament of the Knee.—From the most prominent point of the internal condyle to the inner surface of the inner tibial tuberosity.

External Lateral Ligament.—From the most prominent point of the external condyle to the tip of the head of the fibula.

External Popliteal Nerve.—Behind the tendon of the biceps. Winds forwards round the neck of the fibula.

Posterior Peroneal Intermuscular Septum.—Line from the posterior border of the head of the fibula to the posterior border of the external malleolus. Indicates the line to cut down on the fibula.

Anterior Peroneal Intermuscular Septum.—Line from the anterior border of the head of the fibula to the anterior border of the external malleolus.

Musculocutaneous Nerve (cutaneous part).—Lower half of the above line.

Ankle Joint and Malleoli.—The joint lies ½ in. above the tip of the internal malleolus and 1 in. above that of the external; the external malleolus being ½ in. below and behind the internal.

Peroneal Tubercle.—½ in. below the tip of the external malleolus, the tendon of the peroneus brevis lies above this and that of the peroneus longus below. Each has a synovial sheath, which join above the tubercle, and thence run 1 in. above the outer ankle.

Extensor Brevis Digitorum lies in the interval between the tendons of the peroneus brevis and tertius.

Heads of the Astragalus and Os Calcis are felt in the same angle.

The Head of the Astragalus is also felt in the interval between the tibialis anticus and posticus tendons, and it ought to lie above the line joining the tip of the internal malleolus to the tubercle of the scaphoid.

Sustentaculum Tali is ½ in. below the tip of the internal malleolus. Between the two bony points run the tendons of the tibialis posticus and the flexor longus digitorum; below the sustentaculum runs the flexor longus hallucis.

Tarso-metatarsal Joint of the Great Toe is halfway between the metatarso-phalangeal joint and the tip of the inner malleolus.

The Tubercle of the Scaphoid is halfway between the tarso-metatarsal joint of the great toe and the inner malleolus.

The Astragalo-calcanean Joint on the inner side is just above the sustentaculum tali, and on the outer side midway between the tip of the external malleolus and the peroneal tubercle.

The Calcaneo-cuboid Joint is midway between the peroneal tubercle and the base of the fifth metatarsal bone.

Lower Extremity, ***continued.***

ANTERIOR ANNULAR LIGAMENT :—

1. UPPER PART.—A band two fingers' breadth wide from the anterior margin of the tibia to that of the fibula at their lower ends. Has one synovial sheath, that for the tibialis anticus.
2. LOWER PART.—A Y-shaped band from the os calcis in front of the tip of the external malleolus up to the anterior border of the tibia and across to the inner margin of the instep. Has three synovial sheaths for the tibialis anticus, extensor longus hallucis, and extensor communis with peroneus tertius.

INTERNAL ANNULAR LIGAMENT from the tip of the inner malleolus to the inner side of the posterior process of the os calcis. Has three synovial sheaths beneath it from within outwards for : (1) The tibialis posticus ; (2) The flexor longus digitorum ; (3) The flexor longus hallucis. All of these extend about 1 in. above the malleolus.

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